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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

THE AMERICAN ACADEMY OF PERIODONTOLOGY

Spring Conference

April 25, 26, 27, 1960

Ann Arbor, Michigan

1960 Annual Meeting

October 13, 14, 15, 1960

Mirimar Hotel, Santa Monica, Calif.

BALINT J. ORBAN

Dr. Balint J. Orban, periodontist, oral pathologist, researcher and teacher, is widely known for his many contributions to the art and science of periodontology. He received his medical degrees from the University of Budapest and the University of Vienna and his dental degree from Northwestern University. Dr. Orban's name appeared in the dental literature for the first time in 1923 and since that time he has published 180 scientific papers, many of them in *The Journal of Periodontology*.

Dr. Orban came to this country in 1927 as Professor of Histology and Research at Loyola University in Chicago, Illinois. Two years later he returned to Vienna to teach at the University of Vienna and work with his teacher, Dr. B. Gottlieb. He returned to Chicago in 1938 and since 1948 has been Professor of Periodontics at Loyola University. He has also been a guest lecturer in many dental schools throughout the country. The 5th edition of his excellent book on Oral Histology and Embryology will be released soon. His Atlas of Clinical Pathology of the Oral Mucous Membrane is in its 2nd edition, and his book on Periodontics is now being revised for its 2nd edition.

In 1952 Dr. Orban received the Miller Prize of the Federation Dentaire Internationale; in 1957 he was made a Fellow of the American Academy of Periodontology.

Of the many contributions Dr. Orban has made, probably the most significant are in the role of a teacher. He has a large number of pupils and friends scattered all over this country and the world who both respect and revere him. He is now carrying on a practice limited to oral diagnosis and periodontics with his son, Thomas R. Orban, in Denver, Colorado.

THE JOURNAL OF PERIODONTOLOGY

APRIL, NINETEEN HUNDRED SIXTY

Leukocytes in the Epithelial Attachment

by DANIEL A. GRANT, D.D.S.,* *San Diego, California,* AND BALINT ORBAN, M.D., D.D.S., *Denver, Colorado*

NEUTROPHILE granulocytes are frequently seen in the epithelial attachment. Microscopists have reported seeing these cells in pocket epithelium[1] and in the outer epithelium covering the gingiva.[2] It seems, however, that the possible significance of neutrophile granulocytes in the epithelial attachment has been overlooked in periodontal literature.

Tissue responses in pathosis need careful and detailed study. The data thus obtained must be subjected to thorough scrutiny before valid interpretations can be made. Precise and accurate observations are necessary to enable us to formulate conclusions, to formulate hypotheses and to recognize those assumptions that are implied and are necessary to complete a chain of evidence. The epithelial attachment may serve as a pathway for the penetration of bacterial toxins from the periodontal pocket. Neutrophile granulocytes may be attracted to this tissue and deployed there in response to the invasion by diffusible bacterial products. The observation that neutrophile granulocytes are often found in the epithelial attachment and speculations on the significance of this may add another link to the chain of evidence on inflammatory periodontal disease.

The photomicrographs offered herewith in evidence are of three specimens of human periodontal tissues. The first specimen is an interproximal section obtained from an experiment on gingival curettage. The slide presented is particularly noteworthy since, like a composite, it tells its own story with clarity and completeness. The outstanding feature is the contrasting and clearly demarcated cellular arrangement, where neutrophile granulocytes (polymorphonuclear leukocytes) are found segregated in the epithelial attachment, while plasma cells and lymphocytes are found in the lamina propria.

The keratinized epithelium covering the gingival papilla can be seen in the low power view (Fig. 1) of the slide described. The keratinization ends where

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Histologic material presented in this paper was taken, in part, from research performed in partial fulfillment of requirements for certificate training in periodontology.

Fig. 1. Original magnification 30X. The margin of the gingival papilla is keratinized. The epithelial ridges are long, thin and well defined. The notch in the tooth surface represented the bottom of the pocket at the start of the experiment. Calcified deposits with many micro-organisms are attached to the tooth surface at the notch. The relatively long epithelial attachment is clearly defined. Fiber bundles are embedded in cementum directly apical to the epithelial attachment (EA) and diverge, running laterally and coronally. An inflammatory infiltrate consisting mostly of plasma cells with a few lymphocytes is interspersed between the fiber bundles and seemingly follows the course of the vascular bed. The alveolar crest is visible. The inflammation does not progress into the periodontal ligament.

the epithelium of the gingival margin is continuous with that of the pocket. Calcified deposits and bacterial plaques are adherent to the tooth surface. The space between the soft tissue wall of the pocket and the deposits, was caused by shrinkage during histologic preparation. The outline of the gingival margin so approximates that of the deposits, that it is evident that close contact of gingiva and calculus existed in vivo.

Fig. 2. Original magnification 350X. Filamentous organisms in calculus are attached to the tooth surface. Neutrophile granulocytes are clustered around the micro-organisms. A few shed epithelial cells are found between the organisms and leukocytes.

There is some epithelial proliferation into the lamina propria, which might be a response to the mild inflammation (Fig. 1). The epithelial ridges are long, thin and clearly defined. Connective tissue fibers from the cemental surface apical of the epithelial attachment can be seen extending upward toward the gingival margin. Some fibers are passing horizontally over the alveolar crest. Alongside the epithelial attachment, we can see loose connective tissue with accumulations of inflammatory cells interspersed between the fiber bundles. The inflammation apparently does not progress into the periodontal ligament, but seems to follow the course of the vascular bed. The inflammatory cells in the lamina propria are mostly plasma cells with a few intermingled lymphocytes.

The epithelial attachment is 1 mm. long and is split at the bottom of the pocket (Fig. 1). This might be an artifact or a pre-existent intra-epithelial tear. Within the epithelial attachment itself, neutrophile granulocytes may be found arrayed through the entire length of this tissue. Few can be

Fig. 3. Original magnification 350X. The tear in the epithelial attachment has taken place within the epithelium. Some cells remain attached to the cemental surface. Many neutrophile granulocytes are present in the widened intercellular spaces of the cemental half of the epithelial attachment.

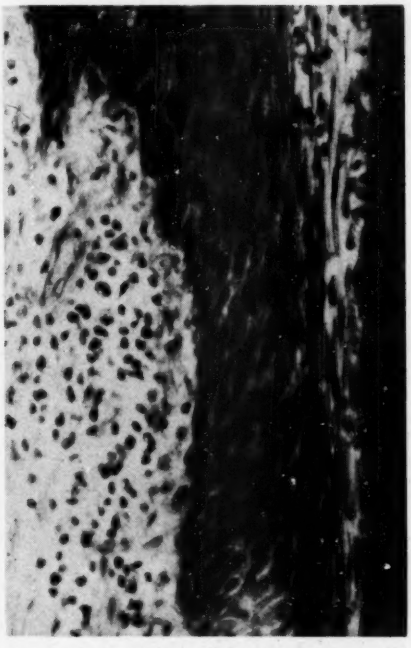

Fig. 4. Original magnification 350X. Some cell destruction is widest in the cemental third of the epithelial attachment. The graduated grouping of neutrophile granulocytes is evident in the widened intercellular spaces of this zone. The infiltrate in the adjacent connective tissue consists almost entirely of plasma cells.

identified in the adjacent lamina propria. Those present were found close to the epithelial attachment. This well defined segregation of neutrophile granulocytes in the epithelial attachment deserves critical evaluation.

Neutrophile granulocytes are clustered around calculus at the bottom of the pocket in the high power magnification of the same slide (Fig. 2). Filamentous organisms are evident in and around the calcified mass. A few shed epithelial cells are prominent between the organisms and leukocytes near the center of the photomicrograph.

The tear in the epithelial attachment is obvious in the next apical sequence (Fig. 3). This higher magnification shows that the separation has taken place within the epithelium and not between tooth and epithelium. Many epithelial cells have been destroyed. Extended cytoplasmic membranes and intercellular bridges form a lattice network background for the numerous neutrophile granulocytes seen in the widened intercellular spaces of the cemental zone of epithelium. A relatively mild inflammatory response is present in the papillary layer of the lamina propria. Some granular leukocytes seem to be migrating from the connective tissue into the epithelial attachment.

As we progress further apically, the graduated grouping of the leukocytes in the cemental third of the epithelium can be seen (Fig. 4). The intercellular spaces in this zone are widened. Some cell destruction is evident. The cemental segment of

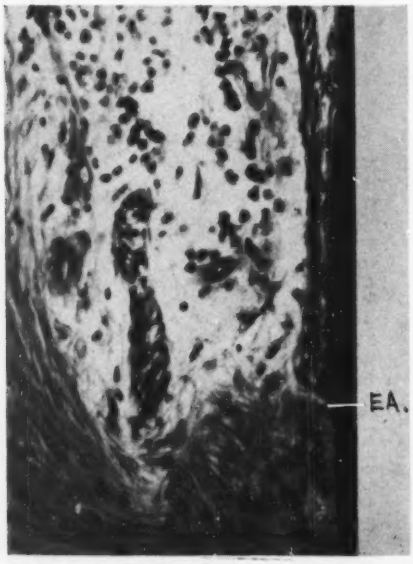

Fig. 5. Original magnification 350X. The epithelial attachment ends (EA.) where dense fiber bundles are embedded in cementum. Neutrophile granulocytes are present in the epithelial attachment to its very end. Adjacent to the epithelial attachment is an area of loose connective tissue with many plasma cells and a few fibroblasts.

the epithelial attachment has long been designated as its weakest portion.[1] This might be due to the action of functional stresses. The inflammatory infiltrate in the connective tissue consists almost entirely of plasma cells. Very few neutrophile granulocytes are present.

The apical end of the epithelial attachment (Fig. 5) consists of relatively few layers of cells. The epithelium ends at a point where dense bundles of connective tissue fibers are embedded in cementum. The fiber bundles extend laterally and then diverge, with some fibers running upward toward the gingival margin. These fibers enclose an area lateral to the epithelial attachment where only a few isolated fibers can be seen. Some clusters of epithelial cells, probably the ends of epithelial ridges, appear as islands in this area. Some fibroblasts and many plasma cells are present in this space of loose connective tissue.

The second specimen is an example of a

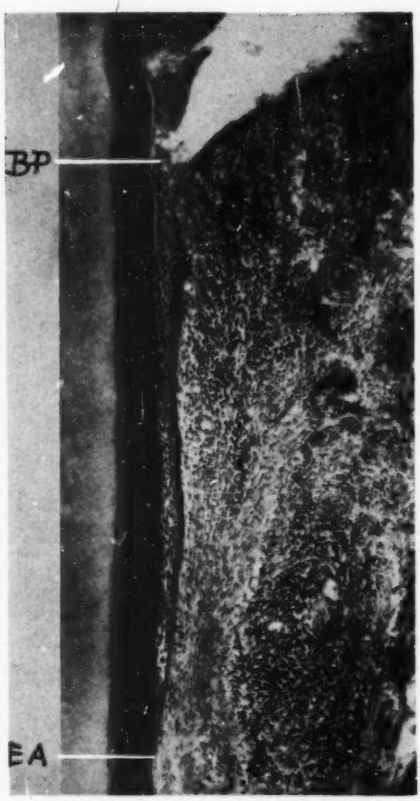

Fig. 6. Original magnification 40X. A severe periodontitis. Calculus and pus are evident at the bottom of the pocket (BP). The pocket epithelium is thin and ulcerated and neutrophile granulocytes are migrating through ulcerations in the epithelium and through the epithelium itself toward deposits in the pocket. The epithelial attachment is 1.1mm long. Apical end of epithelial attachment EA.

severe periodontitis. The low power view of this slide (Fig. 6) shows massive ulceration of the thin pocket epithelium. We can see some poorly defined epithelial ridges that have proliferated into the lamina propria. The subepithelial connective tissue is deeply and densely infiltrated by plasma cells and lymphocytes. Neutrophile granulocytes are migrating through ulcerations in the pocket epithelium and through the epithelium itself toward calculus and bacteria. Suppuration is evident at the surface. The epithelial attachment is 1.1 mm. long, and the widened intercellular spaces con-

Fig. 7. Original magnification 350X. Pus has formed around bacterial plaques. The dental cuticle is attached to the cementum surface.

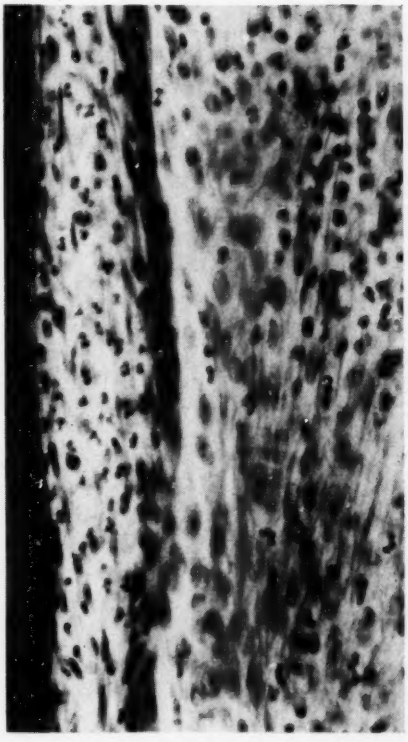

Fig. 8. Original magnification 350X. The central portion of the epithelial attachment is edematous. The widened intercellular spaces are infiltrated by neutrophile granulocytes.

tain many neutrophile granulocytes. In the closely adjacent connective tissue some granular leukocytes are present and may have been migrating toward the epithelium.

The higher magnification of the bottom of the pocket shows neutrophile granulocytes migrating through the epithelium into the pocket forming pus. The dental cuticle can be seen on the surface of the cementum (Fig. 7).

Further apically in the same slide, (Fig. 8), the epithelial attachment is seen to consist of 3 zones. There is a well defined basal cell layer and a narrower zone of cells attached to cementum, which enclose a broad edematous central area. Many cells seem to have been destroyed in this area. Intercellular spaces are very wide and infiltrated by neutrophile granulocytes. The neighboring connective tissue is loose and some neutrophile granulocytes are found between the fiber bundles, probably migrating toward the epithelium.

Neutrophile granulocytes are found in the widened intercellular spaces of the central part of the apical end of the epithelial attachment (Fig. 9). Bacterial products probably penetrate where epithelium is weakest. This corresponds to the zone that is exposed to the greatest functional stresses. We can follow the epithelium as it narrows to single cell width and then ends. Directly apical, connective tissue cells are lying along the cementum. No cementoid is present for about ¼ mm. beneath the epithelial attachment and the subepithelial fiber bun-

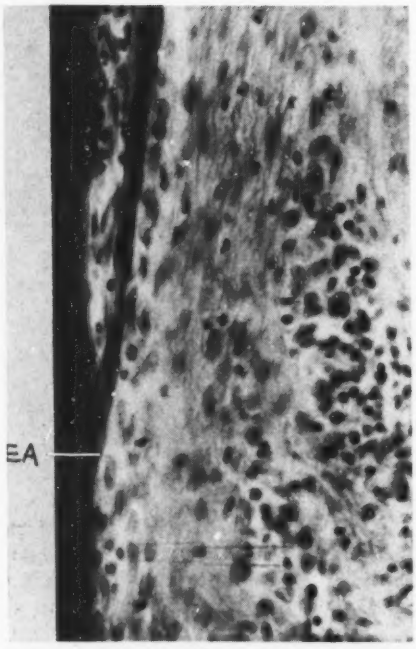

Fig. 9. Original magnification 350X. Neutrophile granulocytes are deployed in the widened intercellular spaces of the epithelial attachment to its apical terminus (EA). Directly apical, connective tissue cells are lying along the cementum.

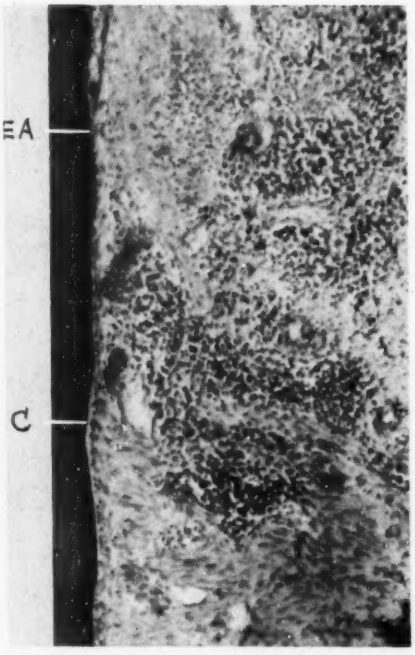

Fig. 10. Original magnification 100X. This lower power magnification of the same specimen shows the apical end of the epithelial attachment (EA). No cementoid is present for about ¼ mm. apical to the epithelial attachment. Apical to this area, cementoid (C) can be seen, deposited on cementum. There is an epithelial rest near this area.

dles have been destroyed (Fig. 10, 11). The lack of cementoid and intact fiber bundles could portend imminent apical proliferation of the epithelial attachment. It could mean that bacterial toxins penetrating via the epithelial attachment (witness the neutrophile granulocytes in the epithelial attachment) have prevented normal deposition of cementum. The continuous deposition of cementum is vital as a barrier to apical proliferation of epithelium.[3] Lateral to the tooth and epithelium, the connective tissue fiber bundles seem to have been destroyed and dense accumulations of plasma cells are found between the fiber remnants. A few granular leukocytes are found close by, coming from venules, probably enroute to the epithelial attachment.

The third specimen is illustrated by a high power photomicrograph (Fig. 12) of an epithelial ridge extending into the lamina propria from the bottom of a pocket. Only the basal cell layers can be recognized. The cells of the prickle cell layer are completely unidentifiable. The widened intercellular spaces are permeated by neutrophile granulocytes. In the connective tissue surrounding the epithelium, dense accumulations of plasma cells can be seen. The distinctive grouping of neutrophile granulocytes and plasma cells cannot be dismissed as a chance occurrence.

The frequent presence of neutrophile granulocytes in the epithelial attachment could be a significant observation. It may offer an additional guidepost to the search for the mechanism of pocket formation. The epithelial attachment might be a pathway for the penetration of diffusible bac-

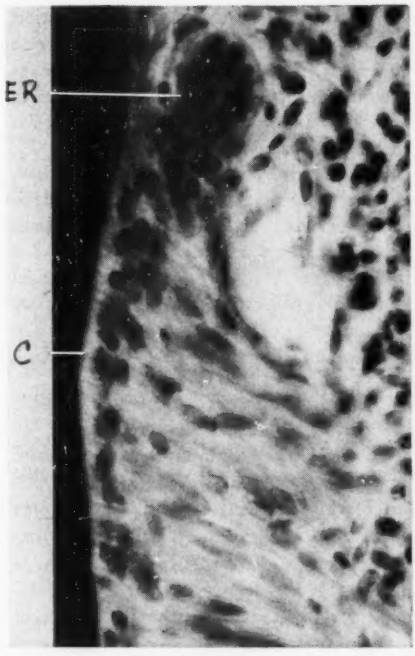

Fig. 11. Original magnification 400X. A high power view showing the area devoid of cementoid and the area apical to this where cementoid (C) has been deposited on cementum. Epithelial rest (ER). Connective tissue fibers are embedded into the newly formed cementum.

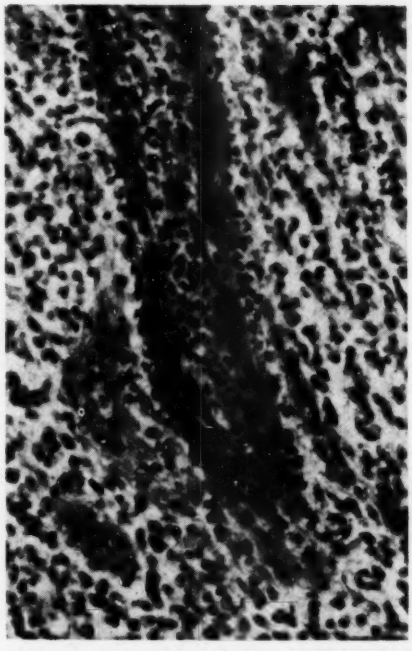

Fig. 12. Original magnification 300X. An epithelial ridge extending into the lamina propria from the bottom of a pocket. The basal cell layers can be distinguished. The cells of the prickle cell layer are unidentifiable. The widened intercellular spaces are permeated by neutrophile granulocytes. The lamina propria contains dense accumulations of plasma cells.

terial products, as shown by the presence of neutrophile granulocytes in its intercellular spaces. An evaluation of this possibility is important with regard to (1) the vitality of cementoblasts subjacent to the epithelial attachment, (2) the destruction of the gingival fiber apparatus in this area and (3) the subsequent apical proliferation of the epithelial attachment.

Recent papers have described the progression from gingivitis to periodontitis as a change largely based on extension of the inflammation along vascular channels to the bone.[4,5] It is presumed that the inflammation spreads from the outer and central portions of the gingival lamina propria toward the cemental surface to destroy the fiber bundles directly beneath the epithelial attachment. This would permit changes to take place that are described as characteristic of periodontitis. If the speculations in this paper prove valid, this view would need further amplification.

While the cellular characteristics of gingival inflammation have been adequately described, attention has focused on the cytological events in the lamina propria. Plasma cells, normally resident in clinically healthy gingiva, increase in numbers in inflammation and become predominant in the characteristic round cell proliferation and infiltrate. Lesser numbers of lymphocytes are usually found, with some macrophages also present. Neutrophile granulocytes have been described as clustered around calculus, bacteria and debris in the pocket. They are found mobilized in ulcerations of pocket epithelium and are teeming in abscesses. The neutrophile granulocytes

sometimes seen in chronically inflamed connective tissue are described as moving from venules toward the pocket.

Orban[6] described polymorphonuclear leukocytes in the epithelium covering chronically inflamed tissue. He speculated that this convergence was due to chemotactic influences from toxins penetrating into the epithelium from the surface or from the products of cellular degeneration. The presence of these leukocytes in the epithelial attachment might be similarly explained on the basis of toxin penetration. Unfortunately, many authorities consider our present knowledge on cytological function in inflammation to be controversial and relatively primitive.[7]

Current and past inquiry into chemotaxis and leukocytic functions has disclosed comparatively little. Chemotaxis is defined as the directional response of a cell or organism to chemical substances in its environment.[8] It is manifested by direction of movement rather than velocity. Neutrophile granulocytes show chemotactic response best. However, particles almost insoluble in plasma have also induced chemotaxis and repeated washings did not decrease the chemotactic effect. McCutcheon[9] believes that electrical charges on particles may stimulate movement, rather than chemical gradients which he felt had not proved demonstrable. He offers as an alternate explanation that particles may differentially adsorb substances from surrounding plasma. The hypothesis does not explain the chemotaxis shown to diverse combinations of polypeptids or the chemotaxis exhibited by cells too far removed from foreign bodies to be affected by electrical charges.

Neutrophile granulocytes are believed to split antigens into soluble fractions.[10] This function could be specifically related to the speculations in this paper. They act as microphagocytes and liberate proteolytic enzymes, notably peptidases, as well as an oxidase.[11] They have a relatively short lifespan and apparently do not reproduce.[7] In the cytological sequence of inflammation, neutrophile granulocytes are the first cells to appear. Where these cells predominate, the inflammation is said to be acute. Menkin[12] claims that these cells are active in an alkaline medium (pH 7.2-7.4). PH is influenced by changes in intermediary carbohydrate metabolism. When glycolytic processes and accumulation of metabolic products affect a drop to pH 6.8-6.9, macrophages which produce enzymes active at this pH, predominate in number. At pH 6.5, all forms of granular leukocytes disintegrate and are removed by phagocytosis.

The neutrophile granulocytes observed in the epithelial attachment could have arrived there because of random movement, or they could have been attracted by diffusion of bacterial products from the pocket. We can presume that bacterial toxins penetrate to the gingival lamina propria[13] where the characteristic inflammatory response has been observed. Except in the case of abscesses, micro-organisms have been only rarely and fleetingly found in the affected tissues. There is considerable circumstantial evidence however, for the causal relation between micro-organisms and gingival inflammation. Micro-organisms are always present in pockets.[14] They are increased in numbers and in percentages of certain types in the presence of inflammatory periodontal disease.[15,16] Organisms whose products are capable of destroying tissue, have been isolated from periodontal pockets.[17] Injection of toxic bacterial products from such micro-organisms has resulted in gingival changes similar to those seen in periodontitis.[18] There is therefore, a presumptive relationship between bacteria and their products and typical periodontal inflammation. In view of such evidence, the possibility of toxin penetration via the epithelial attachment to subjacent and adjacent connective tissue is quite probable. The cytological response seen in the epithelial attachment can be interpreted as a reaction to the presence of diffusible bacterial products.

SUMMARY

1. The presence of neutrophile granulocytes in the epithelial attachment is described.

2. The possible relation of this occurrence in the penetration of diffusible bacterial products through the epithelial attachment is proposed.

3. The epithelial attachment may serve as a pathway for the diffusion of bacterial products to the subjacent and adjacent lamina propria. This would affect (1) the vitality of cementoblasts in the subjacent connective tissue, (2) the integrity of the gingival fiber apparatus and, (3) the subsequent apical proliferation of the epithelial attachment. The postulated series of events may play an important part in the mechanism of pocket formation.

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SOUTHWEST SOCIETY OF PERIODONTISTS

The Southwest Society of Periodontists will hold their fifth annual meeting April 22 and 23, 1960 in Western Hills Motel, Fort Worth, Texas. The subject will be "Etiology and Therapy of Periodontal Disease" and the clinicians will be Dr. E. Cheraskin and Dr. Balint Orban.

For further information write Dr. John H. Swindle, Jr., P.O. Box 3096, Westview Station, Waco, Texas.

Hyperkeratosis of the Oral Mucous Membrane

Associated with Hyperkeratosis Palmo-plantaris Hereditaria

by WILLIAM H. HIATT, B.A., D.D.S. AND BALINT J. ORBAN, M.D., D.D.S., *Denver, Colorado*

THE dental and medical literature contains numerous reports of disturbances involving the keratinization process of the skin, associated with oral lesions. These disturbances in keratinization are hereditary in nature. The oral lesions affect either the periodontium and/or other parts of the oral mucous membrane. According to the literature, involvement of the periodontium is seen most frequently in children and the disease is classified as periodontosis. Such a case was reported in the most recent publication by John I. Ingle[1] where a fourteen-year old boy was observed. E. F. Corson[2] reported the case of a boy eleven years of age and his sister two years of age. E. C. Woods and W. R. G. Wallace[3] observed a case of a ten year old child. They reported dry, scaly skin on the soles of the feet and palms of the hands which had persisted since birth. The nails were dry and striated. S. L. Rosenthal[4] had the opportunity of studying the same individuals reported by Corson, but several years later.

The first authors to describe oral lesions of the periodontosis type associated with hyperkeratosis palmo-plantaris were M. M. Papillon and P. Lefevre[5] who published their findings in 1924. G. Dekker and L. H. Jansen[6] have recently reported periodontosis in a child with congenital hyperkeratosis palmo-plantaris and by complete survey of the literature, have accounted for nineteen cases.

The German literature contains several cases. Hans Schuermann[7] has given a detailed description of various diseases belonging in this group, in his book "Diseases of the Oral Mucous Membrane and Lips." Schuermann seems to agree with Touraine that most of these disturbances are similar in nature, with slight and not significant variations. Touraine suggested the term, "polykeratosis congenita."

As previously mentioned, there are two large groups of lesions of the oral cavity associated with hyperkeratosis palmo-plantaris congenita. That appearing in children is known as the periodontosis syndrome. In the adult, the oral lesions appear as hyperkeratotic manifestations.

There seem to be cases of hyperkeratosis palmo-plantaris hereditaria in which no oral lesions were observed. There is an island, Mljet, in the Adriatic Sea, where intermarriages are frequent and there often occurs a hyperkeratosis of hereditary nature, termed Mal de Meleda. This is a disturbance localized mainly to the palms of the hands and soles of the feet, but oral lesions have not been described. Whether or not the absence of oral lesions is an actual fact or due to an oversight, cannot be ascertained at this time.

Although the most frequent localization of hyperkeratosis in these congenital disturbances is the soles and palms, other areas exposed to mechanical irritation are sometimes involved. In the oral mucous membrane, the palate and cheeks are most frequently affected, although lesions of the tongue have also been noted. In the adult cases, no periodontosis has been reported. Malformations of the teeth, such as fusion, furrows and peg teeth have been observed. Some have given histories of the presence of teeth at birth. There seems to be a tendency to carcinoma of the skin, although some authors have mentioned longevity in these individuals (Schuermann).[7]

Aloys Greither[8] also classifies and discusses these lesions in his text, "Diseases of the Oral Cavity and its Surrounding Areas." The dermatologic literature has

Fig. 1. Extensive callus formation on the hand of white male, age forty, with congenital hyperkeratosis palmo-plantaris.

Fig. 2. The malformed, thick nails of the same patient. (Pachyonychia)

numerous references to congenital hyperkeratotic lesions. Ichthyosis and oral lesions have been observed.

The case reported here is a congenital hyperkeratosis palmo-plantaris associated with hyperkeratosis of the oral mucous membrane. The patient is a white male, forty years of age. The picture of his hand (Fig. 1) shows heavy callus formation in all areas exposed to mechanical irritation. Whenever the patient performs some mechanical function, the callus formation becomes more extensive. The nails are enormously thickened (Fig. 2). The disturbance is known as pachyonychia or hyperonychia. Similar lesions exist on the feet and the callus formations make it difficult for the patient to walk.

The patient, married has one son, age twelve, who shows no lesions. The mother of the patient, age seventy-six, has a similar disturbance. There is no other family history available.

Oral examination of the patient revealed several areas of white patches. Figure 3 shows one area located on the palate. Similar white patches could be seen in the mandibular gingival region, in an edentulous area and in the cheeks. The cheek lesions correspond to bucally tipped second molars,

Fig. 3. Hyperkeratosis, "white patch," of the oral mucosa (palate) of same individual.

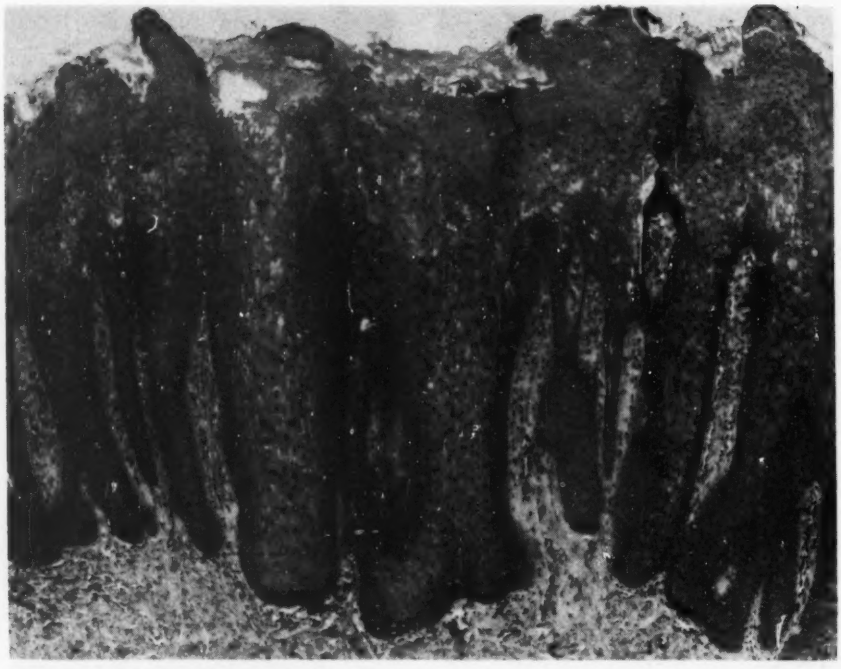

Fig. 4. General view of biopsy of white lesion of the oral mucous membrane (Figure 3). Epithelial ridges elongated, thickened, irregular. Basement membrane intact, very slight inflammatory reaction in the lamina propria. Keratin layer thick, irregular—Keratin and parakeratosis alternate, the granular layer is indefinite.

bilaterally and along the occlusal surfaces of the teeth, extending to the corner of the mouth. The patient smokes a considerable amount.

The microscopic examination of various biopsies from the oral cavity reveals some striking features. The epithelium is thickened, the ridges are elongated and widened. The tips of the ridges are rounded and are sharply separated from the connective tissue. The basal cell layer is regular, with the mitotic activity seemingly increased. The basement membrane is found all along the epithelium and connective tissue border. The keratin layer is thick and irregular; numerous shedding cells are on the surface (Figure 4). In some areas, parakeratosis is in evidence. The keratin layer is separated from the prickle cell layer by a rather wide and irregular granular layer, which appears quite unusual. Under normal conditions, the granular layer consists of several layers of flat cells with elongated dark nuclei and the granules filling the cytoplasm. In cases of hyperkeratosis, the granular layer is often increased in thickness, but still the microscopic features remain the same. In the present case, there are no flattened epithelial cells in the granular layer. The cells are rather irregular, stain lighter and the granules are disseminated (Figure 5). In some areas, the granules extend into the body of the epithelial ridges. The most characteristic feature of this area is the presence of large masses of eosinophilic granules. These are found next to the dark blue keratohyalin granules. Many of the cell nuclei are pyknotic.

The keratohyalin granules stop at the borderline between the prickle cell layer and the granular layer. However, the eosinophilic granular mass extends deep into the

Fig. 5. High magnification of the "granular layer" of Figure 4. The granules are not found in flat cells, but in irregular, rather lightly stained cells. Between the darkly stained keratohyalin granules—some eosinophilic globules can be distinguished.

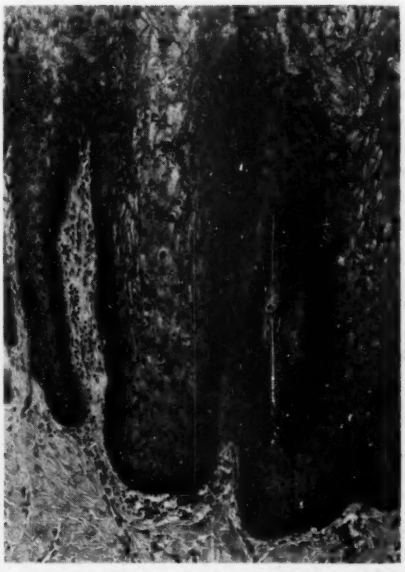

Fig. 6. Higher magnification of some epithelial ridges from Figure 4. In the center of some of the epithelial ridges eosinophilic globules can be followed toward the tip of the ridge.

epithelial ridges in many areas (Figure 6). The eosinophilic globules seem to lie in degenerated cytoplasm of the cells (Figure 7), and are also seen in the intercellular spaces which appear widened in many areas. The eosinophilic material is not seen in the basal cell layer, but seems to be separated from the connective tissue junction by four to five layers of cells. Intercellular bridges are present everywhere in the malphigian layer, except where the eosinophilic globules are heavy.

There is very little inflammation present in the sub-epithelial connective tissue. In a few areas, lymphocytes are seen migrating into the epithelium (Figure 6).

The case has given us some concern as to interpretation of the microscopic findings. Are these white lesions, often called "leukoplakia," better called hyperkeratosis complex or is it a hyperkeratosis simplex?[9] The diagnosis is of great importance. A hyperkeratosis complex lesion should be

Fig. 7. High magnification of the eosinophilic globules seen in the epithelial ridge in Figure 6. Some of the eosinophilic globules seem to be located in the cytoplasm of the cells, others are dispersed in the intercellular spaces, which are enlarged. The basal cells seem not to be affected.

considered a potentially malignant lesion—a precancerous lesion. If this diagnosis is made, all white lesions in the oral cavity of this individual should be eliminated. If, however, the diagnosis, hyperkeratosis simplex, is made, it would not be necessary to remove the lesions. In the present case, after careful consideration, the diagnosis of hyperkeratosis simplex was made and the lesions were not removed. The patient was urgently advised to give up smoking to prevent irritation, and thus aggravation of the lesions. Local application of Vitamin A was prescribed to counteract the tendency to disturbance in the maturation process of the epithelial cells.[10] The patient is kept on observation, and biopsy examination will be repeated.

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FRANK J. KALAS, DC, USN

1912-1959

We have just been informed that Captain Frank J. Kalas, DC, United States Navy, died December 31, 1959 at Camp Pendleton, California, after a prolonged illness. Dr. Kalas was graduated from the University of Louisville School of Dentistry and has been in the Navy since 1941. He was a member of the American Academy of Periodontology and was actively pursuing certification by the American Board of Periodontology at the time of his death.

He is survived by his wife, Mrs. Elizabeth Kalas, and one daughter, Roberta. Interment with full military honors was held at the Arlington National Cemetery.

Ultrasound and Periodontics

by SOL J. EWEN, B.S., D.D.S.,* *Great Neck, New York*

IN the last fifteen years, ultrasonics, that branch of acoustics concerned with sound vibrations in frequency ranges above the audible, has been used therapeutically in dentistry. Most recently periodontics has added this modality to its therapeutic armamentarium. This introduction has met simultaneously with much enthusiasm on one hand and understandable skepticism on the other. Since this biophysical agent is receiving attention both in the literature and at scientific meetings, this article has for its purpose the examination of the physical characteristics, mechanisms of behavior, biologic effects, and some of the potential applications in periodontics.

In 1927, Wood and Loomis published their work on the "Physical and Biologic Effects of High Frequency Sound Waves of Great Intensity."[1] Since that time, in medicine, ultrasound has been used mostly for the treatment of neuromuscular and musculoskeletal ailments. Early investigations of ultrasound dealt mainly with the effects of large doses on animals and animal tissues. In the light of the overenthusiastic reports which have appeared from Europe, with claims of success in the treatment of such diverse diseases as sciatica, malignancies, peptic ulcer, arthritis, etc., understanding, regarding the nature and use of this physical agent as it is particularly applied in dentistry to periodontics, is both timely and justified.

A number of reports concerned with the effects of ultrasound on oral tissues have appeared in the literature. Kennedy, et al.,[2] studying the erupting incisors of very young guinea pigs, report major pathologic changes in the teeth and periodontal soft tissues when subjected to 40 Kc. per second. Burman, et al.,[3] using an ultrasonic generator designed specifically for prophylaxis, found no such extreme changes in the eight human patients treated in their study. They had, however, certain reservations about the tactile sensitivity for the operator and particularly emphasized the poor field of vision due to the water spray. Knapp and Bernier,[4] using fifteen young adult dogs, prepared cavities on maxillary molars. Most of the changes observed in the pulp occurred in the odontoblastic layer and in the pulp tissue proper. Evidence of frank inflammation was absent but vacuolization and edema took place. Most of these changes appeared reversible. The periodontal membrane, the alveolar bone, the temporo-mandibular joint gave no evidence of morphologic alteration. Results to date on work with ultrasound in dentistry have been meager and conflicting. A number of universities and research institutions are consequently active in investigating the effects of ultrasound at 25 to 28 Kc. per second on animals and humans. A review of the physical characteristics and biologic effects of ultrasound in these specific ranges would therefore seem to be in order.

PHYSICAL CHARACTERISTICS

Sonic energy is made up of waves which (a) are propelled longitudinally, (b) require a material medium for transmission, (c) are reflected and also absorbed at the interfaces of dissimilar tissues. It should be understood that ultrasonic vibrations differ from audible sound only in frequency and lie in those ranges above twenty thousand cycles per second. In medical therapeutics the frequencies often employed lie between 800 thousand and one million cycles per second. In dentistry the frequency most commonly employed at this time is 25 thousand cycles per second. The rationale for the use of this particular frequency will be elaborated.

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APPARATUS DESIGN

In 1880 Pierre and Jacques Curie discovered that crystals of many substances subjected to mechanical strains develop electrical charges on their surfaces. Soon, as a natural corollary, it was observed that when crystals of proper size or metals of proper configuration and content were subjected to an alternating electrical field, the crystals or metals so treated vibrated with oscillations of specific frequency and amplitude.

In the ultrasound generator available in dentistry today, such vibratory oscillations (25 Kc.) are produced by utilizing, instead of crystals, a series of metal strips (a "stack") soldered together and tipped off by a solid metal rod. This metallic "strips-rod combination" is placed inside a shockproof and waterproof handle, through which water is circulated and expelled to prevent rises in temperature. This handpiece, filled with flowing water and activated by alternating current, becomes a tool which vibrates 25 thousand times a second in its longitudinal axis, at an amplitude of .001 centimeters. Electrical energy, flowing through such metal rods, may thus be converted into mechanical vibrations. The design of the ultrasonic generator and the size and form of tips of such instruments determine the frequency of these vibrations. Since ultrasonic energy must be transmitted through a propagating medium, the water, in addition to acting as a coolant, as it escapes from the handle becomes a coupling agent between the instrument head and the treatment area. In treatment, the operator can vary only the intensity of output and the duration of application. For example, in using a periodontal tip resembling the traditional curet, six double strokes per unit area of root surface suffice adequately to debride without removing cementum. Should the duration of application be altered by doubling the number of strokes in a given area, detectable amounts of cementum or even dentin may be removed. Such an instrument, therefore, when employed subgingivally, should be used preferably by a dentist or periodontist who is familiar with the clinical and histological extent of the pathosis and the anatomy of the parts to be treated. It is possible to vary the intensity of the dental generator, defined as the watts of power flowing through one square centimeter of area in the direction of the propagated wave, within limits to three gradations—up to 30 watts.

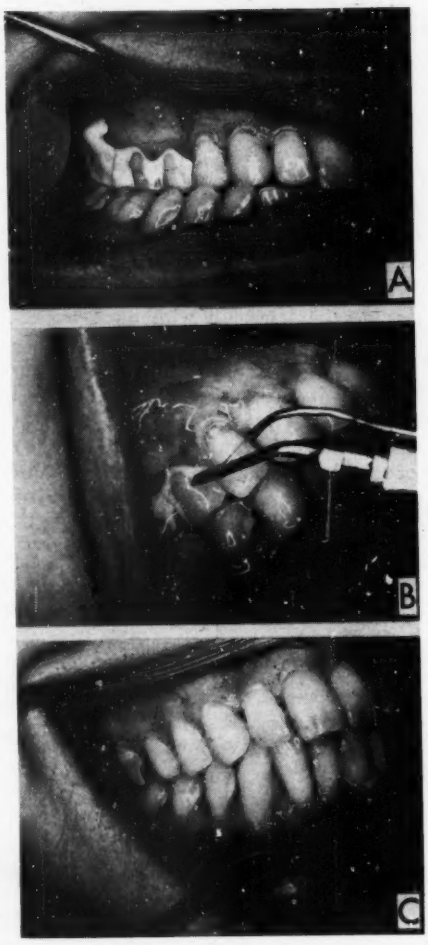

Fig. 1. Ultrasonic scaling. A—Before treatment. B—During initial treatment. C—One week after initial treatment.

Fig. 2. Left—quadrangular grooved ultrasonic probe. Right—ultrasonic Orban file.

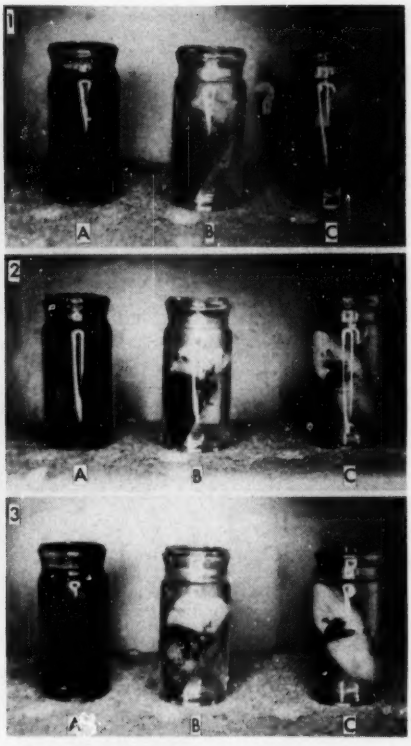

Fig. 3. Ultrasonic dye bleaching. Bottle A—stained tooth in tap water—no manipulation. Bottle B—stained tooth in sodium hypochlorite solution—no manipulation. Bottle C—stained tooth in sodium hypochlorite solution—ultrasonically vibrated.
(Time interval—1B to 3B—five minutes)
(Time interval—1C to 3C—ten seconds)

Fortunately, since the cross-sectional dimension of periodontal instruments is never more than one to four millimeters square, the sound intensity remains very small. However, the apparent adequacy and propitious effects of such dental ultrasonic instruments do not preclude the need for thorough investigation of the intensity limits oral tissues can tolerate. In addition, a knowledge of the basic effects of ultrasound is essential for the development of techniques of application.

BASIC EFFECTS[5]

The primary effects of ultrasound at 25 Kc. (dental apparatus) are: 1. Thermal Effects—sound energy absorbed at tissue junctions takes the form of heat; 2. Mechanical Effects—the physical distortion of the metal stack produces mechanical action in debridement and/or bubbling of the propagating medium (cavitation); and 3. Chemical Effects—the agitation of ultrasonic vibrations releases ions contained in the propagating medium at great speed and intensity.

SECONDARY EFFECTS

The secondary effects of ultrasound are those responses which may be elicited from or produced in a tissue during or following ultrasonic irradiation. These secondary considerations are: 1. Structural heating—(a) vibrations of 25 Kc. by frictional movement can be pressed directly against the tissue to produce coagulation; (b) gentle massage may produce a hyperemia with no tissue destruction, provided that the propagating medium is flowing continuously between the tool and the tissue. 2. Cavitation

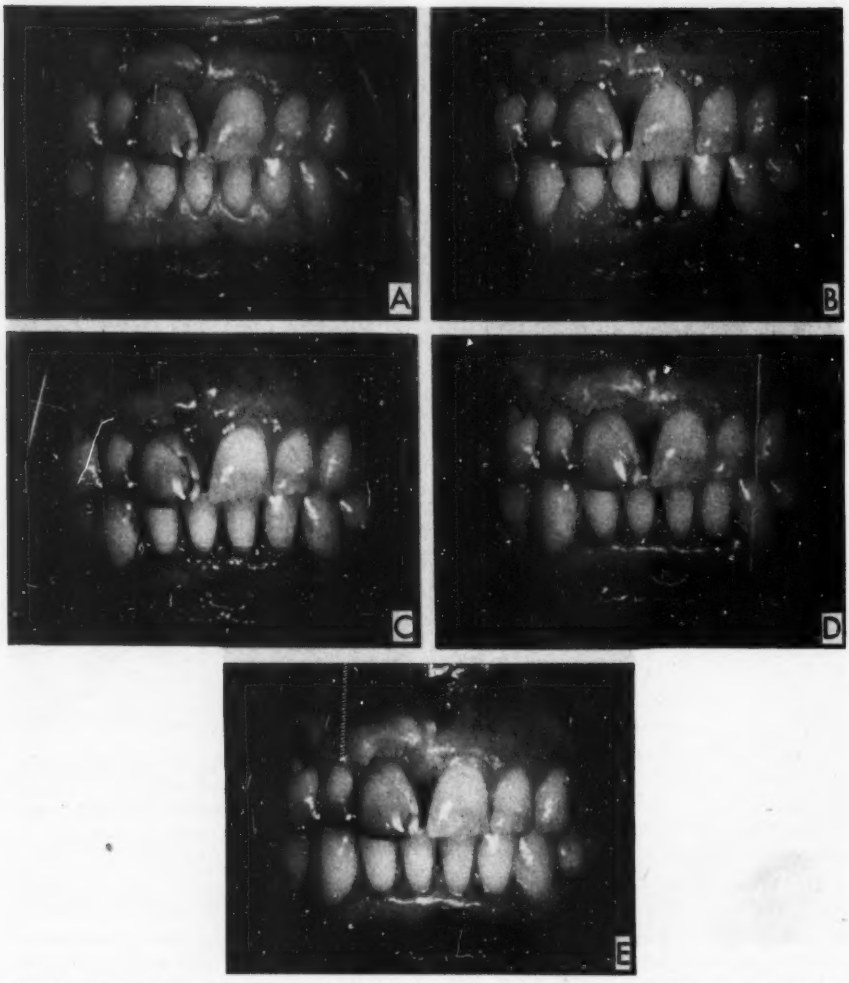

Fig. 4. Ultrasonic labial curettage—six lower anterior teeth. A—Before treatment. B—During first treatment. C—End of first treatment. D—After one week. E—After two weeks.

—ultrasound applied to tissues of high fluid content will evoke bubble formation or degassing within tissue. 3. Mechanical forces—considerable particle force may be released within tissues by ultrasound, the lower the frequency the lower the particle acceleration. 4. Frequency relationships for specific effects—(a) the release of molecular chlorine, iodine, and the anions and cations of Ringer's solution may be valuable in tissue lavage; (b) the suffusion of tissue surfaces with chemicals or heated solutions, e.g., 2-4% sodium fluoride for desensitization of exposed dentin. 5. Tissue surgery--tissues turgid with fluid or frozen solid[6] may be cut with facility with ultrasonic instruments of proper design and frequency. 6. Reduction of gross thermal effects may be achieved by an appropriately pulsating technique by cooling the tissue

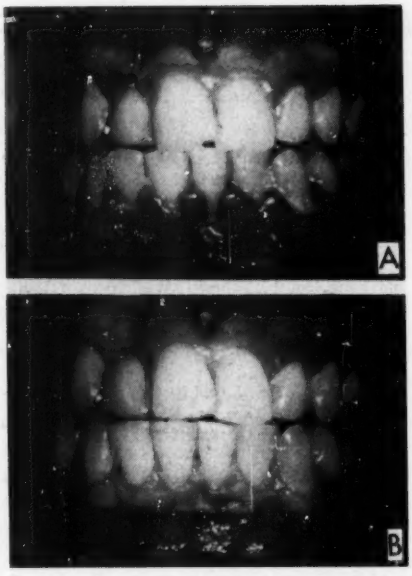

Fig. 5. Edema and swelling—acute marginal gingivitis. A—Before treatment. B—After treatment (one month).

with free-flowing temperature-controlled medium or by moving the instrument tip evenly and smoothly without remaining in any one place for too long a time.

OTHER TISSUE EFFECTS

Research workers have demonstrated that the application of high frequency vibrations has helped improve myalgia and tendon extensibility.[7] Recently in dentistry renewed interest has been manifested in the muscular spasms and pains which accompany temporomandibular syndromes. Might it not be valuable to investigate the effectiveness of treatment of this syndrome with ultrasound?

Research in medicine has shown that scar tissue, particularly that resulting from burns, may be softened following the use of ultrasound.[8] The fibrotic gingiva of chronic gingivitis, being a type of scar tissue, was subjected to ultrasound by the author with similar results to those described.

TECHNICAL ASPECTS OF ULTRASOUND IN PERIODONTICS

In ultrasonic root scaling, it has been demonstrated that satisfactory removal of subgingival calculus is possible (Figure 1). An instrument, quadrangular in cross-section with one side channelized to permit fluid flow to the tip, has been designed by the author (Fig. 2) to permit exact detection of changes on root surfaces. A modified Orban file, thinned down to the thickness of a probe, makes it possible not only to feel the root surfaces but also to file and create fresh cemental or dentinal surfaces. The exact character of the bond of soft tissues to a root ultrasonically scarified has still to be investigated (Figure 2).

The technique for improvement of root surface scaling involves further the use of dye as disclosing agents. Such dyes as gentian violet 2% aqueous, brilliant green, iodo-glycerine, basic fuchsin 1% aqueous, or even Mercurochrome are fine bacteriostatic agents and serve to display the locations of plaque, materia alba, and/or other surface defects. Effective instrumentation may be obtained by using six strokes of the tips per unit area. It is possible to see gross accumulations as well as the finest bits of debris being removed. Treatment is carried out in quadrants and zylocaine 2% is always used to anesthetize the area. Once the quadrant has been painted with the selected dye, salt solution (sodium chloride hypertonic) stored in a pressurized tank is delivered under twelve pounds pressure to the vibrating tool tip. Sodium hypochlorite activated in the same way successfully bleaches out most dyes (Figure 3) and effectively dissolves organic debris which has been dislodged. Proper adjustment of the fluid control knob assists in raising the temperature of the propagating medium to 39 degrees centigrade, enhancing its bacteriostatic action.

Rubbing or pressing a vibrating tool tip against soft tissue coagulates the surface and produces a form of soft tissue curettage. Such curettage may be performed within the crevice or on the buccal or labial

aspects of the gingiva (Figure 4). When ultrasonic vibrations are directed at tissue interfaces, that is, the epithelium-connective tissue junction, they spread laterally lifting off the epithelium. The connective tissue below is dehydrated and the collagen bundles are mechanically pushed apart. The defect thus created in the tissues is a form of coagulated wound. Healing takes place without unusual sequelae. Whether the injury produced is more or less than that produced by hand instruments is being studied. Pulp deaths have never occurred and post-operative tissue edema invariably subsides and resolves to the patient's advantage (Figure 5).

In addition to soft tissue curettage ultrasound may be used for gingival surgery. Periodontal curets sharpened to a razor edge and activated with ultrasonic vibrations are able to excise gingival tissue.

SUMMARY

Physical and biological effects of ultrasonic vibrations at 25 Kc. have been reviewed. Some technical considerations of the clinical applications of ultrasound to root scaling, soft tissue curettage, and surgery in periodontics have been elaborated.

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Beg Your Pardon!

The Editor was somewhat embarrassed to notice that on page 78 of the January 1960 issue of the JOURNAL OF PERIODONTOLOGY Dr. Harold Ray was credited for having presented the Fellowship Certificate to Dr. Harold Leonard. Actually, the correct label should have been, "Dr. Harold Leonard accepts the Merit Award from the American Board of Periodontology from Dr. Raymond E. Johnson."

The Editor is not certain to whom he should apologize for this error so is making a blanket apology to all.

Aids in Evaluating the Dietary Intake of the Periodontal Patient

by J. Lewis Blass, ph.g., d.d.s.* and Jack L. Thaller, d.d.s.,** *New York, N. Y.*

Periodontal disease ensues from a multiplicity of causative agents and shows the characteristics of inflammation and degeneration of the periodontium. No greater importance can be attached to any single etiologic factor, since several combine to produce the pathosis.

One of the etiologic agents is faulty nutritional intake, either in the food itself or its utilization by the body. Miller[1] has demonstrated the significant role that the dietary factors assume in the causation, persistence, and repair of gingival disease. The selection of food as well as its quality and amount occupy an important role in periodontal therapy.

A method is in use at the Department of Periodontia and Oral Medicine,[2] College of Dentistry, New York University, which allows the student, patient, and instructor to cooperate in evaluating the significance of dietary intake in the periodontal treatment plan. This method aids in the study of the dietary intake of the patient and in formulating recommendations for a more satisfactory regimen consistent with basic nutritional principles.

PURPOSE OF THE SIMPLIFIED GUIDE FOR EVALUATING DIET HISTORIES

a) To evaluate the quantity and quality of the patient's food intake relative to the mineral, vitamin, protein and caloric content.

b) To demonstrate graphically the relative discrepancies, inadequacies and excesses in nutritional consumption.

c) To find the patient's daily and weekly caloric total.

d) To show the role of acid-alkali balance in nutrition.

e) To emphasize the need for reduction in refined and demineralized carbohydrate consumption.

f) To indicate the meaning of optimum nutritional intake as differentiated from an adequate diet.

g) To demonstrate the need of roughage, bulk, detergency, and variety in daily food consumption.

h) To serve as a guide for specific dietary recommendations for the patient to compensate for and correct the existing nutritional deficiencies and describe the constituents of an optimum daily diet.

MATERIALS

a) The N.Y.U. simplified guide for evaluating diet histories. (Fig. 1 shows a section of the chart.) This chart is divided into the common food groups with the various subdivisions as follows:

Vegetables
Fruits—Fresh, Canned, Dried, Juices
Milk and Milk Products—Butter, Cheese, Eggs, Cream, Milk
Proteins—Organ Meats, Muscle Meats, Poultry, Preserved Meats
Fish—Canned, Shellfish, Fresh
Salads—Soups and Broths
Fats—Salad Dressings, Sauces and Condiments—Miscellaneous
Sugars—Candies, Syrups, Preserves, Desserts
Grain and Grain Products—Dry Cereals, Cooked
Beverages—Soft Drinks, Beverages, Alcoholic Drinks
Bread and Flour Products—Breads.

Copies of the entire chart may be secured by writing: Dept. of Periodontia and Oral Medicine, New York University, College of Dentistry.

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**Associate Clinical Professor, Dept. of Periodontia and Oral Medicine, College of Dentistry, New York University.

A SIMPLIFIED GUIDE FOR EVALUATING DIET HISTORIES

DEPARTMENT OF PERIODONTIA AND ORAL MEDICINE • NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

FRUITS (Contain vitamins A, B, C minerals etc.)

FRUITS, FRESH (medium piece or portion as noted)

		1 2 3 4 5 6 7	Total
Apple	75		
Apricots (3 medium)	55		
Avocado (4 oz.)	175		
Banana	90		
Berries (8 oz.)	85		
Cherries (8 oz.)	65		
Grapes (8 oz.)	100		
Grapefruit (one-half)	75		
Lemon	20		
Melons (medium portion)	50		
Nectarine	40		
Orange	70		
Peach	45		
Pear	95		
Pineapple (1 slice)	45		
Plum	30		
Tangerine	35		

FRUIT, CANNED – Caloric value 2 oz. drained.

		1 2 3 4 5 6 7	Total
Apple Sauce (sweetened)	45		
Apple Sauce (unsweetened)	25		
Apricots	50		
Fruit Cocktail	45		
Grapefruit (sweetened)	45		
Grapefruit (unsweetened)	20		
Peaches	40		
Pears	40		
Pineapple (2 slices)	95		

FRUIT, DRIED – Caloric value for 1 oz. cooked sweetened.

		1 2 3 4 5 6 7	Total
Apricots	50		
Dates	300		
Figs	55		
Peaches	60		
Prunes	45		
Raisins	50		

FRUIT JUICES – Caloric value for 4 oz.

		1 2 3 4 5 6 7	Total
Apple	65		
Apricot	175		
Grape (bottled)	85		
Grapefruit (sweetened)	65		
Grapefruit (unsweetened)	45		
Grapefruit (fresh)	45		
Orange (sweetened)	70		
Orange (unsweetened)	55		
Orange (fresh)	55		
Pineapple	60		
Prune	85		
Tomato	25		

Fig. 1.

A GOOD DAILY NUTRITIONAL INTAKE

MUST INCLUDE:

1. A 6 oz. glass of juice. Best sources, fresh or frozen, are orange, grapefruit, pineapple, and tomato juice.
2. A 2 oz. portion of natural cheese as cottage, pot, or cheddar.
3. A tossed green salad with a minimum of dressing.
4. Two colored vegetables, fresh or frozen, exclusive of potatoes.
5. Two glasses of milk (May be skimmed milk or buttermilk.)
6. A general portion of muscle meat such as lamb, veal, beef, or poultry.
7. A portion of fish weekly, either fresh, frozen, or canned.
8. A generous portion of organ meat weekly such as liver, sweetbreads, heart, kidney, or spleen.
9. Two eggs any style.
10. Two glasses of water.
11. Fresh fruit, or fresh fruit salad after each meal.

PERMITS A LITTLE OF:

1. Sandwiches to be limited to lunch time and only where necessary. (A hot lunch or salad should be substituted wherever possible.)
2. Bread, 2 to 3 slices, and these are to be of the dark or whole wheat type.
3. No more than three cups of unsweetened coffee daily.
4. Hot cereals; dark if cooked, and sugarless if dry.
5. Butter, preferably one pat.
6. Ice cream without syrup or topping.
7. Unsweetened alcoholic beverages.

MUST EXCLUDE:

1. Cakes, pies, pastries, jams, jellies, chewing gum, candies, puddings, and all other refined carbohydrates are to be restricted or markedly reduced.
2. Flavored carbonated drinks, colas and other fountain drinks. (Juices, tea, milk are substitutes.)
3. Flavored syrups from milk.
4. Fats, gravies, dressings, and fried foods.

Fig. 2.

FOOD DIARY

DATE	BREAKFAST	LUNCH	DINNER	IN-BETWEEN

Patient's Name _____

Fig. 3.

NEW YORK UNIVERSITY, COLLEGE OF DENTISTRY
421 FIRST AVENUE, NEW YORK, N.Y.
DEPARTMENT OF PERIODONTIA AND ORAL MEDICINE

The foods you eat have much to do with your general health and especially the health of the gums and teeth. To help in planning your diet, please record in detail the foods and drink you have consumed for a period of one week. Your dentist will provide you with an evaluation of your diet as part of your treatment and will make recommendations for your specific needs.

INSTRUCTIONS FOR THE USE OF THE FOOD DIARY

1. In the proper squares on the reverse side, immediately after each meal record all the food and drink you have had.
2. In order to help you in the treatment of your condition, it is important that you continue your routine diet during this week.
3. List all the items in detail; do not leave out the smallest item, such as gum, colas, cakes, etc., in the columns provided.
4. List the type and quantity of the foods you eat using such abbreviations as:

T - tablespoonful	cn - canned	fr - fried	gl - glass
t - teaspoonful	s - sugar	sl - slice	
c - cup	br - broiled	pl - plain	

5. Be sure to record the use of sugar, ketchup, fried foods and gravy. Be sure to state the amount of each food eaten, such as:

INCORRECT	CORRECT
Juice	4 oz. Fresh Orange Juice
Sandwich American Cheese	American Cheese 2 sl. white bread lettuce, tomato, butter
Coffee	1c coffee 2 ts with 2 T milk
Dessert	1c peaches cn
Vegetables	1/2c peas cn
Potato	1 medium baked potato

6. Please write clearly:

Personal History

TYPE OF BRUSH USED.....DENTIFRICE.....MOUTHWASH.....FREQUENCY.....
METHOD OF BRUSHING (up and down, circle, across etc.).....FREQUENCY.....
MEDICATIONS OR VITAMINS.....WHY.....
LAXATIVES.....FREQUENCY.....
WEIGHT.....GAIN.....LOSS.....PERIOD OF TIME.....
MEALS: REGULAR.....IRREGULAR.....FAMILY.....ALONE.....APPETITE: GOOD.....BAD.....
RAPIDITY OF EATING: FAST.....SLOW.....SALT INTAKE: LIGHT.....MODERATE.....HIGH.....
CIGARETTES.....LIQUID INTAKE GLASSES PER DAY: WATER.....COFFEE.....TEA.....JUICE.....
FOOD DISLIKES.....
FOOD ALLERGIES.....
FOOD PREFERRED.....

Fig. 4.

b) The N.Y.U. specific dietary recommendation form. (Fig. 2) On the reverse side of Fig. 2 ample space is provided for the dentist or student to make specific dietary recommendations for the patient which have been determined by him after evaluating the patient's dietary intake for the trial period.

1. The guide is used after the availability of a food diary for one week. (Fig. 3 & 4)

2. The entries on the food diary are transferred to the guide sheet. Each constituent food item is inserted in the proper square on the chart by means of an X.

3. The calorie totals for the individual food units and total calorie intake for the trial period are tallied. This is done by multiplying the calorie value for the food unit by the number of times it appears on the chart. This value is inserted in the column to the right of the item. The caloric values for the nutritional groups are secured by adding the individual caloric totals of each group and inserting the number in the column at the extreme right of the page.

4. The total calories consumed during the trial period is arrived at by adding all the calorie totals at the extreme right of the page. The daily calorie value is secured by dividing the number of days of the test period.

5. Varicolored pencils may be used for recording the values of carbohydrates, protein, fat and other food groups to make discussion of nutritional deficiencies or excesses more graphic.

6. Specific vitamin or other therapy may be recommended consonant with good therapeutics.

7. In the presence of existing physical ailments, especially of a gastro-intestinal or metabolic nature, consultation with the patient's physician is necessary.

CONCLUSION

1. A modified guide for evaluating diet histories and the use of a dietary recommendation form used in the Department of Periodontia, College of Dentistry, New York University is presented.

2. The value and purpose of the forms employed is shown.

3. A method for dietary evaluation that leads to better education of patients and improves results in periodontal treatment is demonstrated.

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10 Years of Tooth-Mobility Measurements

by HANS R. MÜHLEMANN, *Zurich, Switzerland*

DISEASES of the tooth-supporting structures necessarily influence the solidity of anchorage or the degree of tooth-mobility (TM). Increased mobility or loosening of teeth is a cardinal symptom of periodontal disease. The severity of periodontal disease cannot be correctly appraised without assaying the magnitude of mobility.

Comparably, anemia may be tentatively diagnosed by the appearance of a person. However, a correct diagnosis of blood disease is only possible by measuring the amount of hemoglobin, iron, etc. in the blood.

This year, we are celebrating a decennial of research and routine use of TM-measurements or periodontometry. It is an opportunity to review critically our experience; some of our basic investigations on TM having been previously reported in this journal (1-5). Periodontometry (6), as practiced at this school, is simple and reproducible, but the method is not ideal. It is definitely an improvement over the older, extraoral measuring method of Elbrecht (1939,[7]), Werner, (1942,[8]), Cross (1951,[9]), but further developments may be expected. The vitrometer of Beyeler and Dreyfus (1947,[10]; 1949,[11]) and of Manly et al. (1951,[12]) and the electromagnetic mobility recorder of Lundqvist (13), which are based on different working principles, have not been developed beyond the experimental stage.

Reports concerning investigations with our periodontometer have been made by Breitschwerdt (14), Forsberg and Häggland in Stockholm (15, 16, 17), Franke and Lindemann in Germany (18), Egli (19) and Meier (20) in Switzerland, and Karlson in the U. S. A. (21).

Two measuring devices have been developed:

1. The macroperiodontometer (22, 23)
2. The microperiodontometer (23, 24)

The principle of mobility recording is similar in both instruments (Fig. 1). They differ in size and accuracy only.

The *macroperiodontometer* (Fig. 2) is designed to be used for measurements of horizontal mobility of incisors, cuspids, and first cuspids, preferably in the upper jaw. A dial indicator is mounted on an impression tray and adapted with the indicator's pointer to the labial or buccal surfaces of the teeth to be measured. Measurements are in hundredths of millimeters.* The labio-palatal or palato-labial deflections of teeth are made by a known force using a *force meter* (Fig. 3). Macroperiodontometry is easily learned and its reproducibility is high (23).

The *microperiodontometer* (24, Fig. 4) is a very small dial indicator permitting mobility measurements of all teeth. The method is tedious, difficult to learn, less reproducible (23) and only indicated when the mobility of molars has to be determined.

Periodontometry has also been used in reversed application (24, 25) for the determination of the magnitude of oral muscle forces transmitted to the crowns of teeth.

MECHANISM OF TOOTH-MOBILITY

The greater the horizontal forces which are applied for 1 to 2 seconds to the crowns in the palatal or labial direction, the greater the TM or excursion of the crown. However, the increase in crown excursion is not linear to the increase in forces deflecting the crowns. Fig. 5 represents the degree of mobility in relation to the amount of forces applied. Initially, with forces up to 100 grams, a relatively

*Mobility in this paper will always be expressed in hundredths of millimeters.

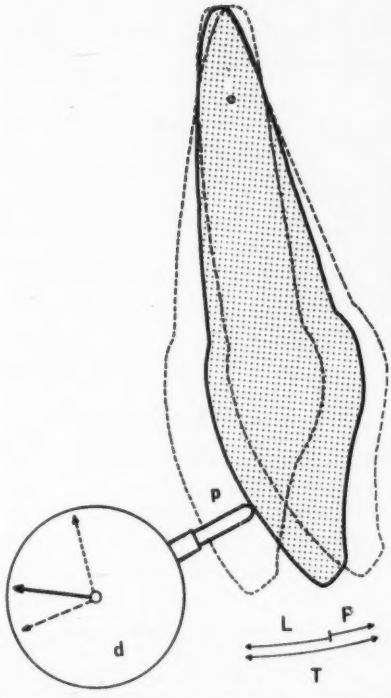


Fig. 1. Principle of mobility measurements. d = dial indicator p = pointer L = Labial deflection of crown P = Palatal deflection of crown T = Total excursion.

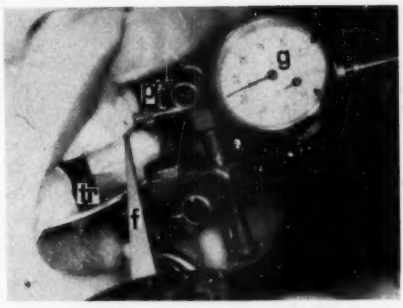


Fig. 2. Macroperiodontometer g = measuring gauge attached to modified impression tray (tr) p = pointer of gauge f = pointer of force meter which causes crown deflections.

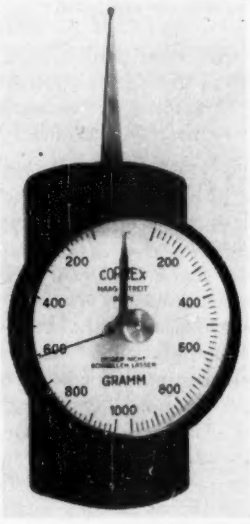


Fig. 3. Force meter with diamond pointer.

large total excursion (T) results. This mobility range has been called the *initial TM* (ITM) (22, 6, 1). Force increments from 100 grams upwards produce smaller mobility increments indicating greater resistance to the imposed force. This is the range of the *secondary TM* (STM).

The *initial TM* (T_{100}) corresponds to an intra-alveolar displacement of the root associated with a preparation of the alveolodental fibers on the tension side for functional action (3). Measurements of TM with 100 gram forces therefore yield information on the functional state of the periodontal membrane. Initial TM is much greater in the presence of loosely and poorly structured membranes than in membranes having dense and distinct collagenous fibers. Initial TM is absent in ankylosed teeth (6), as intraalveolar root

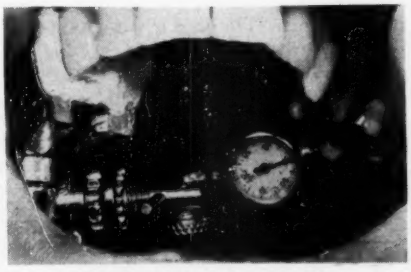


Fig. 4. Microperiodontometer.

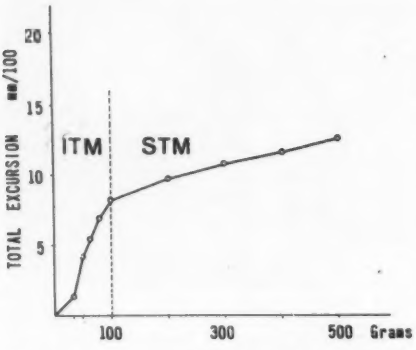


Fig. 5. Magnitude of labio-palatinal mobility (mm/100) of upper incisor (♀, 20 years old, normal conditions) in relation to forces (grams) applied to the crown. ITM = Range of initial TM. STM = Range of secondary TM.

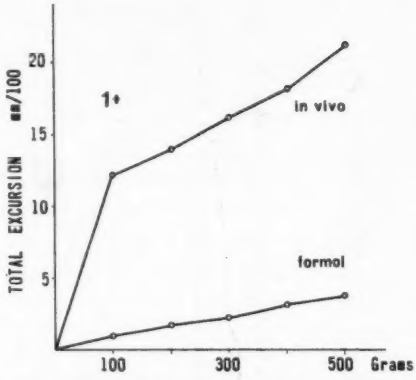


Fig 6. Loss of "initial TM" by formol treatment. Upper curve: In vivo mobility of upper incisor in Rhesus monkey. Lower curve: Mobility of incisor after exarticulation of jaw and formol treatment.

displacement is not possible. Similarly, the experimental chemical alteration of periodontal structure by denaturation by formalin or heat (4) on freshly exarticulated jaws is associated with a loss of initial TM (Fig. 6).

In the range of secondary TM, i.e. deflection of crowns with horizontal forces above 100 grams, the whole periodontium is reversibly distorted (Fig. 5,[3]). Therefore, measurements with 500 grams for example yield information on the elasticity of the periodontium as a whole. For routine work, it is recommended to determine the secondary TM with 500 grams (T_{500}) (23). An international standardization of the forces used is highly desirable, otherwise comparison of data is greatly impaired or even impossible.

THE RATIO T_{100}/T_{500}

In the past, TM on our patients has always been measured with 100 grams and 500 grams. The ratio T_{100}/T_{500} was considered to be of diagnostic value. An analysis of our data has shown that this is untrue. The average ratio is about the same at different mobility levels (Table 1) and is not significantly changed by periodontal treatment (Table 2). From this, it may be concluded that an increase or decrease of TM is produced by changes occurring simultaneously in the periodontal membrane and in the alveolar bone, which together form a functional unit. Changes in the periodontal membrane are not limited to the membrane but have repercussions on the whole periodontium. For this reason, there is no loss of diagnostic information by periodontometry if only one force, 500 grams, is used. However, it is advisable to determine the mobility of several teeth within an individual.

TABLE I

Ratio of T_{100}/T_{500} at increasing mobility levels in 470 teeth of 66 persons

Level of T_{500} (mm/100)	*0–9.9*	*10–19.9*	*20–29.9*	*30–39.9*
No. Teeth	111	211	92	56
T_{100}/T_{500}*	.60±.12	.60±.14	.61±.07	.65±.10

* Mean ± Standard Deviation

TABLE II
Ratio of T_{100}/T_{500} before and after treatment in 74 teeth of 26 patients showing a therapeutic mobility decrease

Classes of Mobility Decrease (T_{500})	*2.0 to 4.9*	*5.0 to 9.9*	*Over 10.0*
Mean Mobility Decrease	3.28	7.16	14.3
No. Teeth	32	26	16
Ratio Before Treatment	.605	.582	.627
Ratio After Treatment	.584	.563	.620
Decrease of Ratio	.021 ± .079*	.018 ± .104	.007 ± .082

* Mean ± Standard Deviation

NORMAL MOBILITY

The range of the most frequent mobility values (T_{500}) in adults free of periodontal disease are assembled for different tooth types in Table 3. Mobility is greater in children and young adults. It is slightly greater in females, and it increases considerably during pregnancy (6). Moreover, mobility values fluctuate during a 24-hour period, the lowest being in the evening and the highest in the morning, just after awakening (26, 27, 18).

T_{500}-values greater than the upper range indicated in Table 3 are suspect of being pathological.

A pathological increase in mobility is due to:

a. *qualitative changes* in the biophysical state of periodontal tissues, or

b. *loss or destruction* of periodontal structures (quantitative factor).

QUALITATIVE CHANGES

Alterations in the biophysical state of periodontal structures are due primarily to local circulatory disturbances which are associated with changes in tissue oxygenation and nutrition.

Such alterations of the local periodontal metabolism may be artificially produced by compressing the periodontal membrane by means of interdental ligatures (28) or orthodontic appliances (29, 5). The increase of the TM measured is considerable and is probably due to anemic states of periodontal membrane areas. The figures shown in Table 4 are for interdental ligatures and in Table 5 for orthodontic appliances.

A similar disturbance of tissue oxygenation is produced by bruxism. Repeated states of hypoxia may be one of the conditions responsible for the increase in mobility in bruxism patients (28, 30). Changes

TABLE III
Ranges of most frequent tooth mobility values of different types of teeth

Teeth	T_{500} *mm/100*
Incisors	10–12
Canines	5– 9
Premolars	8–10
Molars	4– 8

TABLE IV
Average increase of tooth mobility (T_{100}, T_{500} in mm/100) through interdental wire ligature applied between lower incisors and left in situ for four hours

	No. Teeth	Before Trauma	After Trauma	Increase	Percentage Increase
T_{100}	34	6.75	9.24	2.48	36.7
T_{500}	34	9.28	12.58	3.30	35.6

in TM have made possible the diagnosis of bruxism hitherto unaware to the patient (31).

Modifications of capillary tone and blood circulation are always found during pregnancy. Concomitantly, there is an increase in mobility due to physiochemical changes of the supporting structures (6). Such changes have been measured by Gans et al. (32).

Similar qualitative periodontal alterations are produced by occlusal trauma. Increased mobility is one of the cardinal symptoms of abnormal forces transmitted to teeth (23, 33, 34, 35). Patients with longlasting unilateral masticatory function show a greater increase in mobility on the hypofunctional side than on the hyperfunctional side (35). The reestablishment of normal function in cases of functional disharmonies is associated with a pronounced decrease of mobility (Fig. 9,[36]).

Circulatory disturbances are always associated with inflammatory reactions of periodontal tissues to marginal irritants. The successful treatment of periodontal inflammation in cases of periodontitis is followed by a decrease of mobility. This has been shown in a study (37) which included 2 groups, A and B. Mobility was measured before and 33 days after (group A), or 60 days after (group B) the mere removal of marginal irritants. Mobility decreased in both groups approximately by 10%.

The very first (initial) symptom of periodontosis is an increase of mobility. Periodontosis is often due to an inherent local circulatory disturbance as will be shown later.

QUANTITATIVE CHANGES

Quantitative changes of the supporting structures are the consequence of an increased rate of resorption of bone or a decreased apposition rate.

Periodontal atrophy may be very pronounced without mobility being increased (see case H. T.).

It is evident that changes in the biophysical quality of periodontal structures contribute more towards a mobility increase than a loss of periodontal structures. Mobility is definitely increased in teeth with deep pockets (14, 38). However, in a statistical analysis, the correlation between TM and maximal periodontal bone loss was rather poor ($r = +0.56$) (38) (Fig. 7). This has also been shown in an experiment where artificial labial pockets of 6 to 7 mm were created (Fig. 8) (39) by removing part of the labial alveolar bone. Artificial pocket creation produced an immediate increase of T_{500} for 2 from 16.0 to 18.5 (15%), and for 1 from

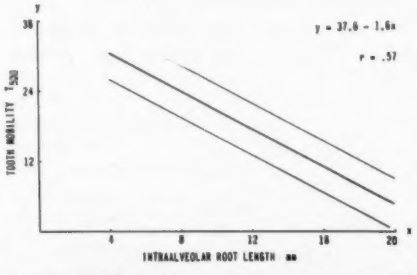
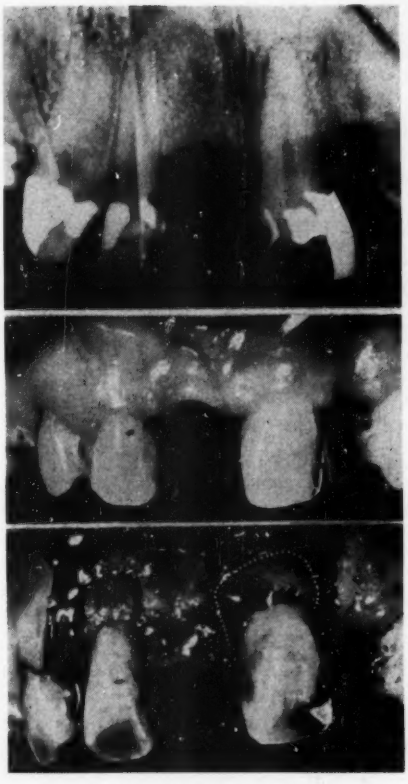


Fig. 7. Relationship between tooth mobility (T_{500}) and length of intraalveolar portion of single rooted teeth.



before	16.0	43.5
after	18.5	51.75

Fig. 8. Case H. F. Tooth mobility (T_{800}) before and after removal of part of labial alveolar bone. The line of bone separation has been drawn for the upper left central, but not for the upper right lateral, where it is easily visible.

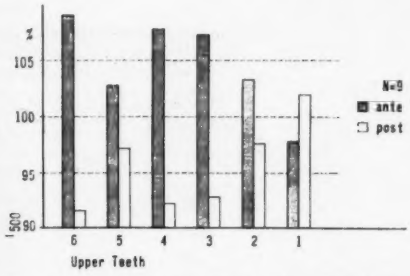


Fig. 9. Tooth mobility (T_{800}) before (black column) and after (empty column) selective grinding in 9 patients with unilateral masticatory function. The base line "100%" corresponds to the average value of all measurements of each group of both homologous teeth. The columns show the mean percentage deviations from the base line for both the hypo- and hyperfunctional side.

43.5 to 51.75 (18%). These mobility increments, obviously due to a quantitative change, are modest when compared to those being produced by qualitative modifications such as the example summarized in Table 4. .

TM CHANGES AND THEIR INTERPRETATION

The great sensitivity of periodontometry permits the quantitative assessment (1) of the influence of systemic factors, (2) of local factors on periodontal tissues and therefore (3) of the effect of various therapeutic procedures. An adequate and generally comprehensible presentation of mobility data involves problems inherent in all attempts to express degrees of disease

TABLE V

Increases of tooth mobility (T_{800}) during the application of different types of forces (orthodontic appliances)

Force Originated by	*Appliance*	*Most Frequent Increases*
spring action	labial arch on Hawley retainer	30
rubber elastic	modified Hawley retainer	40
screw action	modified Hawley retainer	13
masticatory musculature	inclined plane	20
lip musculature	oral screen	7

TABLE VI

Two methods of expressing therapeutic tooth mobility decreases as percent reductions

Mobility before Therapy	*Mobility Decrease*			
	$X_1-X_2=5$		$X_1-X_2=20$	
	%	corr. %*	%	corr. %
100	5.0	5.3	20.0	21.3
60	8.3	9.3	33.3	37.0
30	16.7	20.8	66.6	83.3
15	33.3	55.5	—	—

X_1 T_{500} before therapy X_2 T_{500} after therapy

* $\frac{X_1-X_2}{X_1-a} \cdot 100$ a = 6, lowest value occurring in incisors.

or health arithmetically. If for example the average mobility has decreased during the period of observation in group A from 50 to 45, the difference of 5 is meaningless to those not familiar with periodontometry. Therefore, percentages have been used to express mobility changes. However, this has serious shortcomings.

From the previous example, one may say that mobility decreased by an average of 10%. However, an average 10% decrease would also be calculated if mobility had changed from 10 to 9. Those familiar with periodontometry will readily agree that a therapeutic average decrease of mobility from 10 to 9 (10%) is a far greater achievement than an average decrease from 50 to 45, which is also 10%. Therefore, a corrective formula is suggested for those preferring percentage values. Usually, percentages are calculated by the formula $\frac{x_1 - x_2}{x_1} \cdot 100$, x_1 equaling T_{500} before, $x_2 = T_{500}$ after therapy. If the lowest mobility value (a)* corresponding to any type of tooth is subtracted from x_1, we arrive at the corrected percentage formula: $\frac{x_1 - x_2}{x_1 - a} \cdot 100$.

The role played by the value "a" in calculating percentage figures is presented in Table 6. If mobility decreases from 15 to 10, the uncorrected percentage reduction would amount to 33.3%. Since a mobility decrease from 15 to 10 means a return to healthy conditions ($T_{500} = 15$), it is an understatement to speak of a mobility reduction of 33.3%. By using the corrected formula, we find a 55.5% improvement of the state of health.

*The following figures were assigned to "a" for the different types of teeth:

upper incisors a = 6
upper cuspids a = 3.5
upper bicuspids a = 5
upper molars a = 3

TABLE VII

Patient H. T., male, age 27. Severe periodontal atrophy. Unchanged mobility (T_{500}) of upper front teeth in 1951 and 1958

Teeth	3	2	*1*	+	*1*	2	3
12.11.51	8¼	13½	13		12½	14¼	9½
18.11.58	10	17¼	12½		12¼	13¾	8½

TABLE VIII

Patient I. F., female, age 25. Beginning periodontosis. Pathomobility in May 1957 and further increase of mobility between May 1957 and October 1958 despite of systemic and local treatment

Teeth		3	2	*1*	+	*1*	2	3
May 1957	Before Treatment	9¾	15½	14¾		13	14¾	8¾
July 1957	After Vitamine E Therapy	9¼	16	13		13¼	14¼	8½
October 1957	End of local Treatment							
October 1958		10	15½	16		15	15	10

DIAGNOSTIC VALUE OF PERIODONTOMETRY

The diagnostic value of periodontometry will be discussed in presenting two case histories.

1. *Case H. T.*, male, age 27.

In 1951, the patient was alerted because of his "receding gums". The clinical condition at that time is shown in Fig. 10A, the radiological status in Fig. 11A, and the TM-data in Table 7. Medical history as well as functional analysis of occlusion were negative. A diagnosis of noninflammatory periodontal atrophy was made.

The association of almost normal mobility values with definite bone atrophy was positively interpreted in a prognosis of periodontal health. The patient had no periodontal treatment whatsoever during the next seven years. In 1958, periodontal atrophy and recession were less apparent due to inflammatory gingival enlargements caused by marginal irritation. However, mobility values were still similar to those found in 1951, thus confirming the correctness of the diagnosis 7 years ago (Table 7). Only the upper right lateral incisor showed an increase in mobility associated with the formation of a pathological pocket.

2. *Case I. F.*, female, age 25.

During her first visit in May 1957, Miss F. complained about the migration of upper incisors. The clinical and radiological periodontal conditions are shown in Figs. 12 and 13. There were no pathological pockets and no atrophy of periodontal structures present. However, mobility values of the upper front teeth were abnormally increased (Table 8). Therefore a diagnosis of "beginning periodontosis" was made. The pathological mobility was explained by

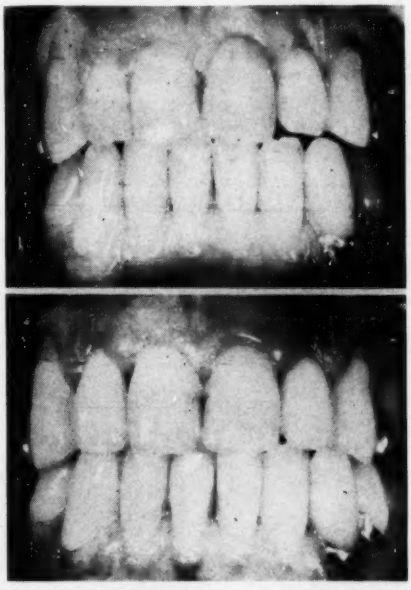


Fig. 10. A) Patient H. T., male, age 27. Clinical status in 1951: Periodontal atrophy. B) Status in 1958: Slight gingivitis. No periodontal treatment during interval 1951-1958.

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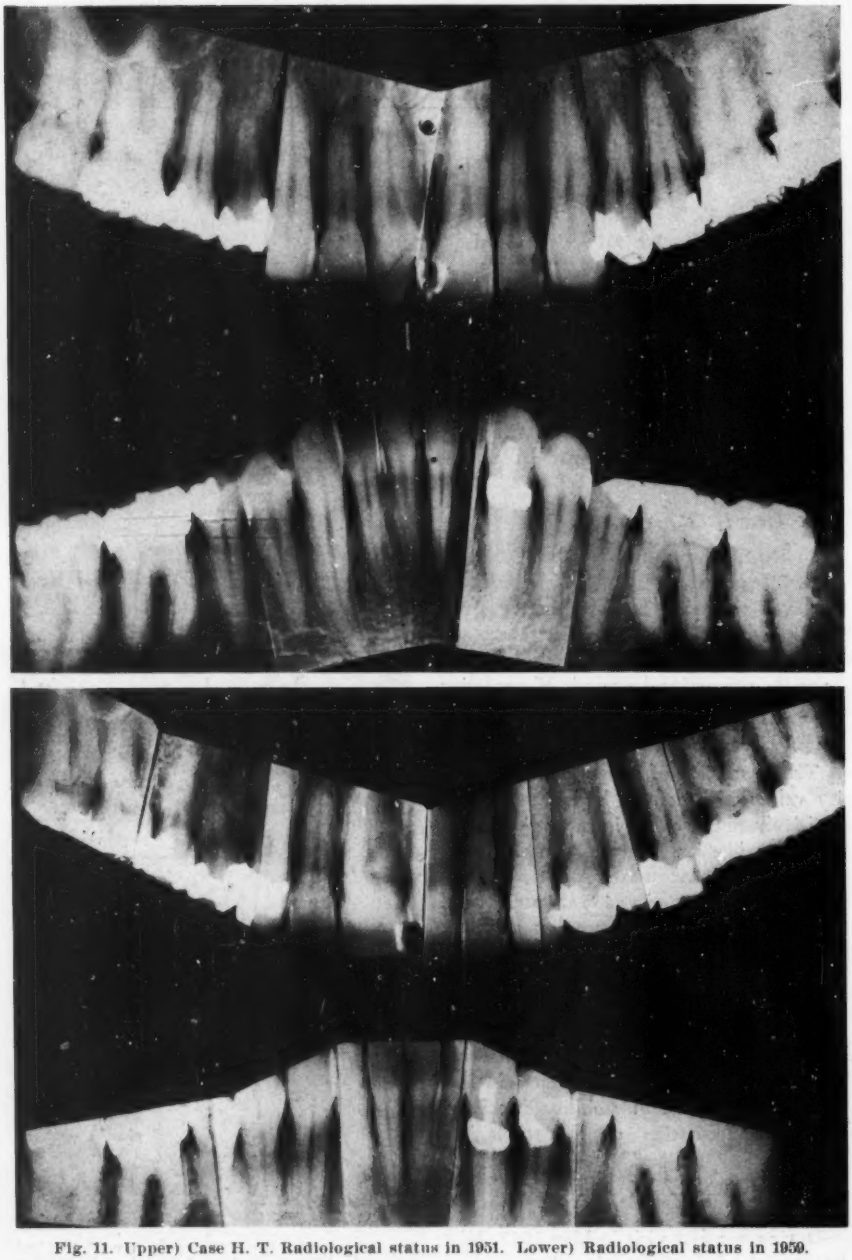


Fig. 11. Upper) Case H. T. Radiological status in 1951. Lower) Radiological status in 1959.

Fig. 12. Case I. F., female, age 25. Periodontosis. Beginning migration of upper incisors. Slight gingivitis.

qualitative changes of periodontal structures due to periodontal circulatory disturbances. The patient complained about cold hands and feet. Experimental rewarming curves made

1) at the skin surface of hands exposed to ice water during 2 minutes and
2) within the gingival sulcus, exposed first to room temperature and afterwards to an ice bag for ½ minute

were abnormal. This is shown in Figs. 14 and 15. Rewarming of hands and gingival tissue was greatly disturbed and slower in comparison to controls.

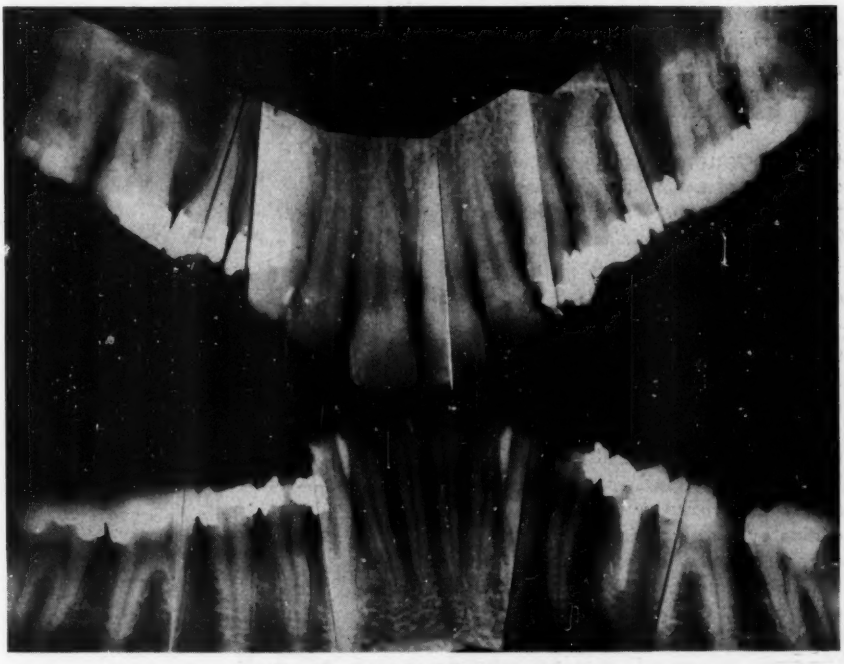
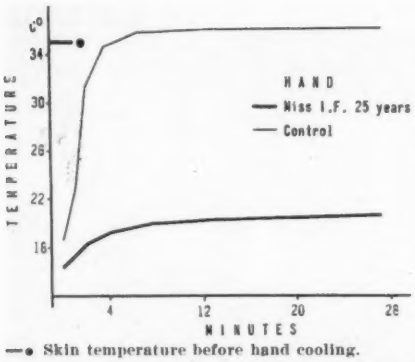


Fig. 13. Case I. F. Radiologic status.



—• Skin temperature before hand cooling.

Fig. 14. Rewarming curves of hands exposed to ice water for 2 minutes in control and patient I. F. Absence of temperature increase of skin during observation period.

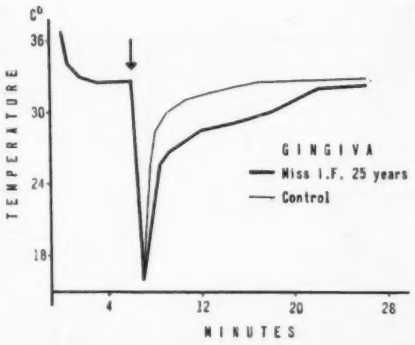


Fig. 15. Rewarming curves of marginal gingivae exposed to an ice bag during ½ minute. Initial temperature decrease due to room temperature and rapid fall (↓) during application of ice. Rewarming delayed in patient I. F. when compared with control.

A further observation of the patient proved the diagnosis to be correct. Despite systemic (vitamin E) and local treatment, mobility values increased during the consecutive one year observation period by 11%, which is in marked contrast to the previously presented case H. T., which showed no significant mobility changes in a 7 years interval.

EFFECT OF PERIODONTAL TREATMENT ON TOOTH MOBILITY

TM measurements permit an objective evaluation of the effect of various therapeutic procedures. We have shown that injections of biostimulines (placenta extracts) have no effect on tooth mobility (37).

Tooth mobility changes after local treatment have been described several times (37, 40) and recently, using new material, in detail by Wüst (41). Part of the data are presented in Fig. 16. Periodontal treatment consisted, for all patients, in the removal of marginal irritants, curettage and/or gingivectomy, equilibration of occlusion by selective grinding, correction of minor anomalies in tooth position, and fastidious home care (Charters' method in most cases). Group A (13 patients) received removable splints in the upper jaw. Group B (8 patients) was not splinted.

The schedule of treatment and mobility measurements was the following:

1. Diagnosis, first mobility determination.

2. Local treatment during 2 months, insertion of splint in group A.

3. Mobility measurements after an additional 2 months.

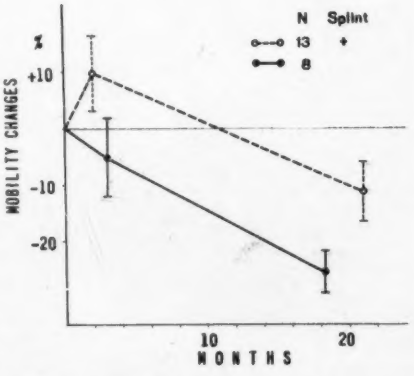


Fig. 16. Percentage changes in tooth mobility two months after end of local therapy and after an additional 16 to 19 months' observation period (corr. %).

O-------O Group A, wearing removable splint since end of local therapy.
●———● Group B, no splinting.

4. Mobility measurements after 20 months.

It can be seen from Fig. 16 that mobility decreased approximately 11% in non-splinted cases, whereas it increased in splinted cases. Only 20 months later was there a definite decrease in mobility both in group A and B.

CONCLUSIONS AND SUMMARY

A review of our 10-year-experience with tooth mobility measurements is given. Pierodontometry is used in Zurich in the clinical routine and for research. The various modes of application are discussed. Studies concerning the effect of systemic or local—pathological or therapeutic—influences on the periodontal structures and investigations of abnormal masticatory function and occlusal trauma greatly benefit from reliable mobility measurements. It should be realized by those interested in bone and connective tissue metabolism that the tooth offers a unique opportunity for the in vivo evaluation of biophysical alterations of supporting tissues.

We would like to express our appreciation to Dr. Charles Eugster, D.D.S., for correcting the English manuscript.

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DR. JAMES P. HOLLERS HONORED

Professional associates of Dr. James P. Hollers paid high tribute to him at a testimonial dinner in San Antonio recently, when nearly 500 men attended the event which was sponsored by the San Antonio District Dental Society.

Dr. Hollers has made many important contributions to San Antonio; he was influential in the building of the Alamo Stadium and very active in the San Antonio Medical Foundation. He is currently the President of the Texas Dental Association and President of the American Academy of Periodontology. Rumor has it that he is to be a candidate for the office of President-elect of the American Dental Association in 1962.

The Normal Glucose Tolerance Pattern: The Development of Blood Glucose Normality by an Analysis of Oral Symptoms*

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THERE is still considerable debate as to what should be regarded as the *normal* glucose tolerance pattern. The presently accepted range has been developed by an analysis of *presumably* healthy individuals. This report is designed to analyze the glucose tolerance pattern in a group of dental patients with and without *oral symptoms* which are ordinarily recognized as being common findings associated with diabetes mellitus.

REVIEW OF THE LITERATURE

In order to appreciate the design and results of this study, it is necessary to review briefly: (1) the common oral symptoms associated with diabetes mellitus, and (2) the present basis for the so-called normal glucose tolerance pattern.

ORAL SYMPTOMS

The three most commonly reported oral symptoms believed to be associated with diabetes mellitus are: (1) *gingival tenderness*, (2) *xerostomia*, and (3) *stomatopyrosis*.

Gingival Tenderness: Frequent mention has been made of this complaint in the literature but no figures are cited.[1-9] Sheridan et al[3] observed occasional to constant gingival tenderness in 62 percent of their dental patients with diabetes mellitus in contrast to the appearance of this symptom in 32 percent of individuals with normal glucose tolerance patterns.

Xerostomia: Several reports have appeared in the literature which indicate a relationship between dry mouth and diabetes mellitus. Sheppard[10] examined a group of 100 diabetic patients including 12 under the age of 21 and 4 above 70 years of age. He divided the diabetic individuals into 3 categories which he designated as mild, moderate, and severe diabetes mellitus. However, no mention is made in his report as to the criteria for his severity classification. He reported xerostomia as constant or almost so in 6 percent of the cases, occasionally present in 20 percent, and rarely observed in 8 percent of his series. Banks[11] and Amies and Park,[1] without citing specific figures, also reported the relatively frequent association of dry mouth and diabetes mellitus. Barach[2] observed xerostomia in 75 percent of his series of 336 diabetic patients. Sheridan et al[3] studied 100 dental patients. They observed 8 percent of the diabetic individuals with constant xerostomia and 46 percent with occasional dry mouth.

It is clear from these limited reports that there appears to be some relationship between xerostomia and diabetes mellitus. The various investigators have, however, reported different percentages. This may be due, at least in part, to the difficulty in quantitating this particular symptom.

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There is also the possibility that some or all of the differences may be the result of the variations in the standards for a diagnosis of diabetes mellitus.

Stomatopyrosis: This particular symptom has been described as being associated with diabetes mellitus by Sheppard.[10] However, no specific details were described in his publication. Sheridan and his coworkers[8] showed that 35 percent of their dental patients who proved to have diabetes mellitus also reported occasional burning mouth in contrast to 8 percent of the nondiabetic individuals with the same complaint.

TABLE 1
Age Distribution

Age groups	*Total number of patients*
0- 9	1
10-19	9
20-29	19
30-39	27
40-49	19
50-59	17
60-69	8
Total	100

NORMAL GLUCOSE TOLERANCE PATTERN

There are two general approaches for establishing normal values:[12] (1) *statistical analysis*, and (2) *physiologic analysis*.

Statistical Analysis: In general the values for the normal glucose tolerance pattern have been derived from an examination of *presumably* healthy individuals. In most cases, subjects have been regarded as well if *relatively* asymptomatic. Mosenthal and Barry[13] have published a set of values which are now generally regarded as the normal glucose tolerance pattern. Their conclusions have been based upon the findings in 50 ambulatory hospital workers. These investigators suggest that the normal glucose tolerance test is represented by a fasting venous glucose level of 100 or less

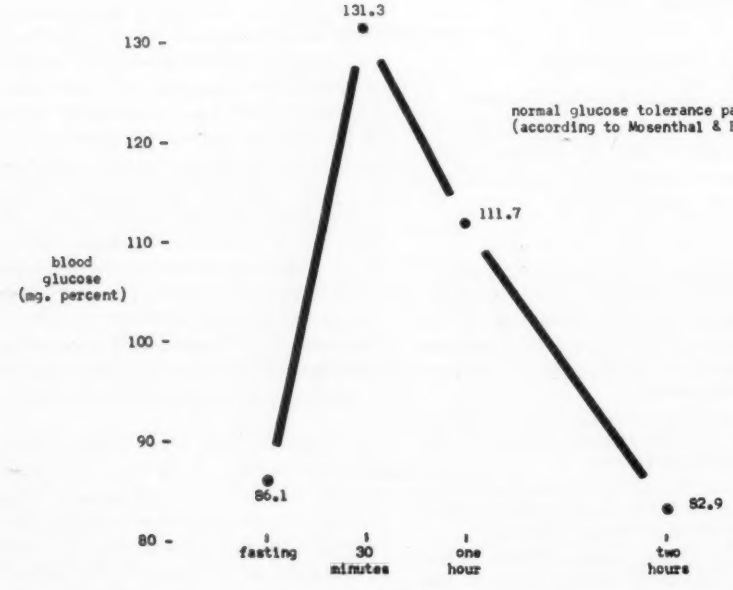

Figure 1

TABLE 2
Statistical Comparison of Mean Differences

		Blood glucose differences				
		Fasting	*30 minutes*	*One hour*	*Two hours*	*Three hours*
Mosenthal and Barry Study versus Alabama Study	Mean difference	3.4	6.4	7.2	18.1	
	P	>.200	>.200	>.200	<.005	
Gingival tenderness versus no gingival tenderness	Mean difference	5.4	5.0	4.5	14.3	10.5
	P	.500	.500	.500	>.200	>.200
Xerostomia versus no xerostomia	Mean difference	2.6	9.4	14.2	17.7	16.0
	P	.500	>.200	>.200	>.100	>.100
Burning mouth versus no burning mouth	Mean difference	1.2	19.4	25.1	23.4	2.4
	P	.500	<.025	<.025	<.025	.500
Burning mouth-gingival tenderness versus no burning mouth-no gingival tenderness	Mean difference	2.4	16.9	11.4	20.3	0.3
	P	.500	>.200	.500	>.100	.500
Dry mouth-gingival tenderness versus no dry mouth-no gingival tenderness	Mean difference	5.4	10.9	12.1	23.6	17.9
	P	>.400	>.200	>.200	>.050	>.050
Dry mouth-burning mouth versus no dry mouth-no burning mouth	Mean difference	3.8	23.9	36.0	43.4	21.2
	P	>.400	<.025	<.025	<.001	>.100
Dry mouth-burning mouth-gingival tenderness versus no dry mouth-no burning mouth-no gingival tenderness	Mean difference	8.3	36.3	32.3	41.4	12.8
	P	<.010	<.010	>.200	<.001	>.400

mg. percent, no venous glucose level greater than 150 mg. percent, and a return of the blood glucose level to 100 or less mg. percent at the end of two hours. Figure 1 shows the specific mean values obtained by Mosenthal and Barry in their study.

Physiologic Analysis: There is no published report available which attempts to analyze the normal glucose tolerance pattern on the basis of the absence or presence of oral symptoms which are commonly associated with diabetes mellitus.

METHOD OF INVESTIGATION

One hundred dental patients were studied in the Section on Oral Medicine at the University of Alabama School of Dentistry. As far as possible, the subjects were selected at random. Table 1 shows the age distribution of the group. It can be observed that the pattern approaches a typical unimodal curve. Included in this study were 22 males and 78 females.

Each subject was carefully questioned regarding possible xerostomia, stomatopyrosis, and gingival tenderness. The scoring was made on the basis of a three-point scale. If the patient reported negatively, 0 was assigned. When the individual indicated occasional dry or burning mouth or gingival tenderness, then 1 was recorded. Two was utilized to indicate the constant presence of the particular symptom. Thus a completely asymptomatic patient was given a total score of 0; a patient with all three complaints constantly was assigned 6.

A true glucose tolerance test was performed on each patient according to the methods of Somogyi[14,15] and Nelson.[16] No preparatory diet was recommended except for complete fasting for 12 hours prior to the laboratory examination. Complete details regarding the method of investigation

TABLE 3
Comparison of Dry Mouth and Age

Age groups	No dry mouth		Occasional dry mouth		Constant dry mouth		Total number of patients	Percentage of patients
	Number	Percentage	Number	Percentage	Number	Percentage		
0- 9	1	100					1	100
10-19	8	89	1	11			9	100
20-29	13	68	6	32			19	100
30-39	18	67	8	29	1	4	27	100
40-49	9	47	10	53			19	100
50-59	11	61	7	39			18	100
60-69	4	57	1	15	2	28	7	100

are available in the publication by Sheridan and his group.[3]

RESULTS

The findings will be reported in two ways: (1) general characteristics of the entire group, and (2) subgroup analysis.

GENERAL CHARACTERISTICS

Figure 1 pictorially represents the suggested normal glucose tolerance pattern derived by Mosenthal and Barry from a study of 50 presumably healthy hospital workers. Figure 2 summarizes the data derived from a study of 100 routine dental patients. Figure 3 is a composite graphic analysis of the findings in the 50 hospital workers in the Mosenthal and Barry series and the 100 dental patients in the Alabama study. The small mean difference at the fasting level, thirty and sixty minute

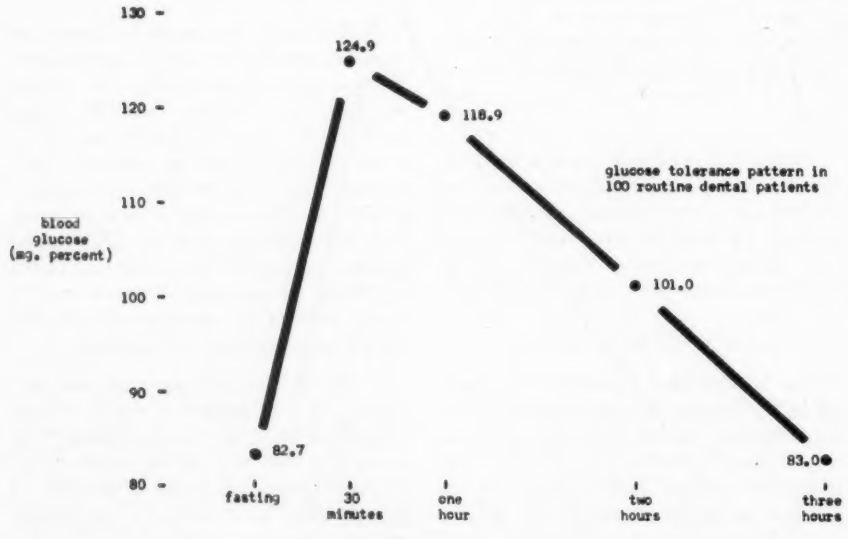

Figure 2

points in the two groups is not statistically significant ($P > .200$) as outlined in Table 2. The only point at which there is a statistically significant difference of the means is at two hours ($P < .005$).

SUBGROUP ANALYSIS

The three symptoms studied will be analyzed as: (1) *independent*, and (2) *combination groups*.

Single Factors: Three symptoms were analyzed: (1) *gingival tenderness*, (2) *dry mouth*, and (3) *stomatopyrosis*.

Gingival Tenderness: The 100 patients in the Alabama series were divided into two groups: (1) those who reported gingival tenderness, and (2) those without gingival tenderness. Forty-six percent of the cases were symptomatic, 54 percent free of this particular symptom. Figure 4 shows the glucose tolerance patterns obtained from these two groups on the basis of the presence or absence of this one symptom. Five points deserve comment. First, the glucose tolerance pattern of the symptomatic group suggests a decrease in tolerance compared with the asymptomatic group. Secondly, in the main, the mean differences are small throughout. Thirdly, the greatest mean difference occurred at the two-hour point. Fourthly, the three-hour blood glucose in the asymptomatic group is below the initial level. In contrast, the glucose level is higher at three hours than initially in the group with gingival tenderness. Fifthly, it is clear from Table 2 that there is no statistically significant difference between these two groups.

Dry Mouth: The 100 patients in the Alabama series were divided into two groups: (1) those who reported xerostomia, and (2) those who were asymptomatic in this regard. Sixty-four percent of the individuals proved to be xerostomia-free; 36 percent complained with dry mouth. Figure 5 shows the glucose tolerance patterns obtained from these two groups on the basis of the presence or absence of this one symptom. Five points are worthy of mention. First, the xerostomia group shows a relatively decreased glucose tolerance pattern. Secondly, the smallest differences appear at the fasting levels. In the main, the mean differences increase at the various temporal points. Thirdly,

TABLE 4

Comparison of Dry Mouth and Age

Age groups	*No dry mouth*		*Occasional dry mouth*		*Constant dry mouth*	
	Number of patients	*Percentage of patients*	*Number of patients*	*Percentage of patients*	*Number of patients*	*Percentage of patients*
0- 9	1	1				
10-19	8	13	1	2		
20-29	13	20	6	18		
30-39	18	27	8	25	1	33
40-49	9	14	10	33		
50-59	11	17	7	20		
60-69	4	8	1	2	2	67
Total number of patients	64		33		3	
Percentage of patients		100		100		100

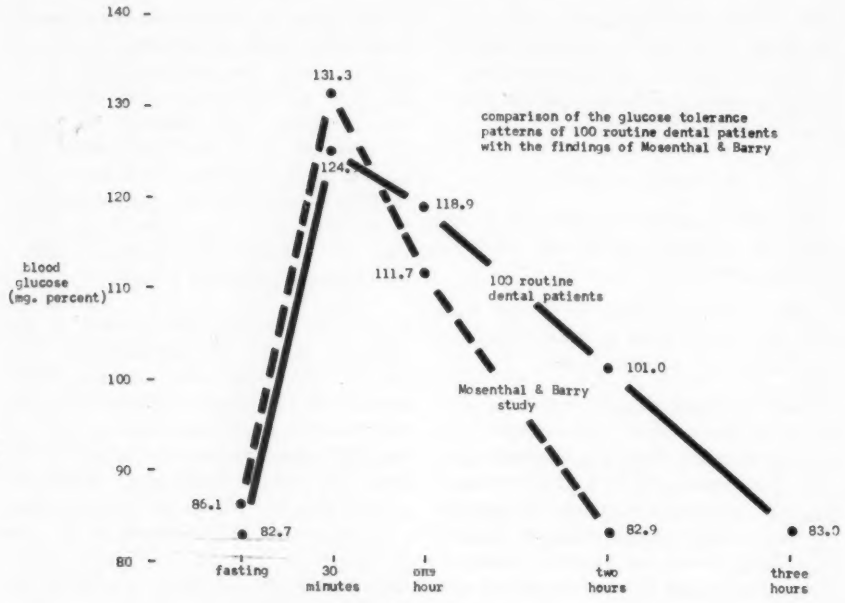

Figure 3

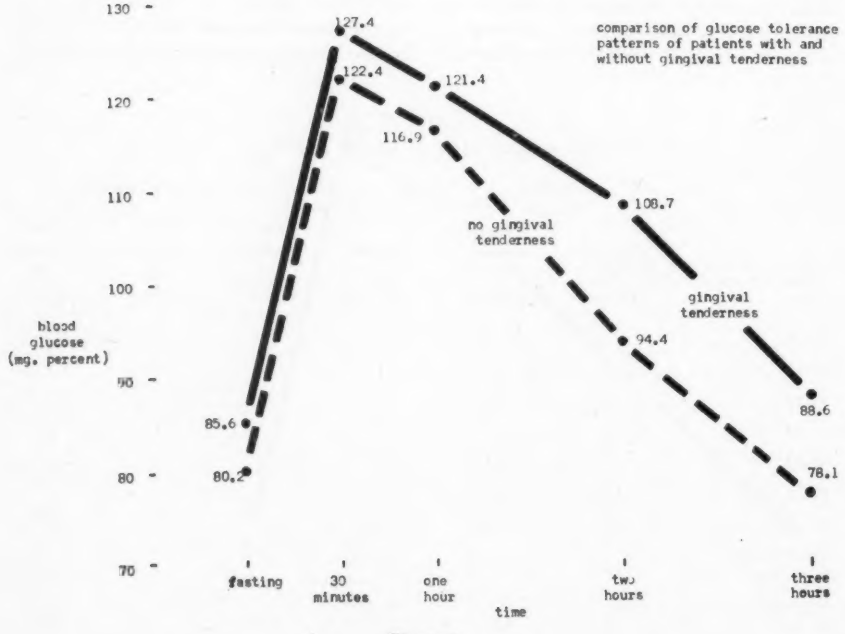

Figure 4

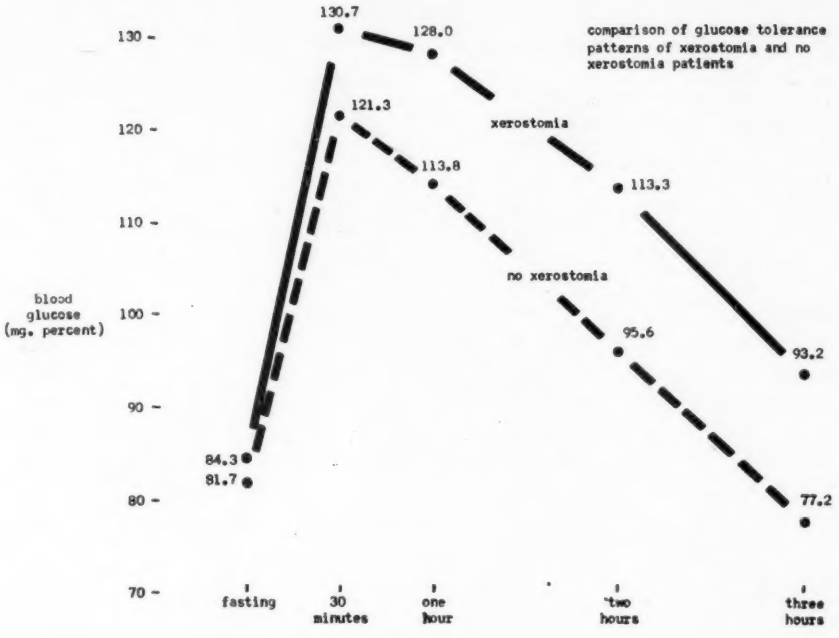

Figure 5

the greatest mean difference is at two hours. Fourthly, the 3-hour level in the xerostomia-free group is below the initial glucose level. In contrast, the 3-hour glucose level in the xerostomia group is higher than that observed initially. Fifthly, it is clear from Table 2 that there is no statistically significant difference between these two groups.

Stomatopyrosis: The 100 patients in the series were divided into two groups: (1) those who reported burning mouth, and (2) those who were asymptomatic in this regard. Eighty-six percent were without this symptom; 14 percent complained with burning mouth. Figure 6 shows the glucose tolerance patterns obtained from these two groups on the basis of the presence or absence of this one symptom. Five points deserve special mention. First, the burning mouth group show a relatively decreased glucose tolerance pattern. Secondly, the smallest mean differences appear at the fasting levels. Thirdly, the greatest mean differences occur at the one-hour, then the two-hour, then the thirty minute points. Fourthly, the three-hour blood glucose level in the symptomatic group is slightly higher than initially. The fasting and three-hour blood glucose levels are essentially the same in the asymptomatic group. Fifth and last, Table 2 shows statistically significant mean differences ($P < .025$) at 30 and 60 minutes and two hours.

Combination Factors: Three independent symptoms have already been considered. In this section, an analysis will be given of the four combinations of these symptoms: (1) burning mouth and gingival tenderness, (2) dry mouth and gingival tenderness, (3) dry and burning mouth, and (4) dry and burning mouth and gingival tenderness.

Burning Mouth-Gingival Tenderness: Fifty-five patients reported the presence (7 patients) or absence (48 patients) of

TABLE 5

Comparison of Burning Mouth and Age

Age groups	No burning mouth		Occasional burning mouth		Total	
	Number of patients	Percentage of patients	Number of patients	Percentage of patients	Total number of patients	Percentage of patients
0- 9	1	100			1	100
10-19	9	100			9	100
20-29	19	100			19	100
30-39	24	89	3	11	27	100
40-49	16	84	3	16	19	100
50-59	14	77	4	23	18	100
60-69	3	43	4	57	7	100

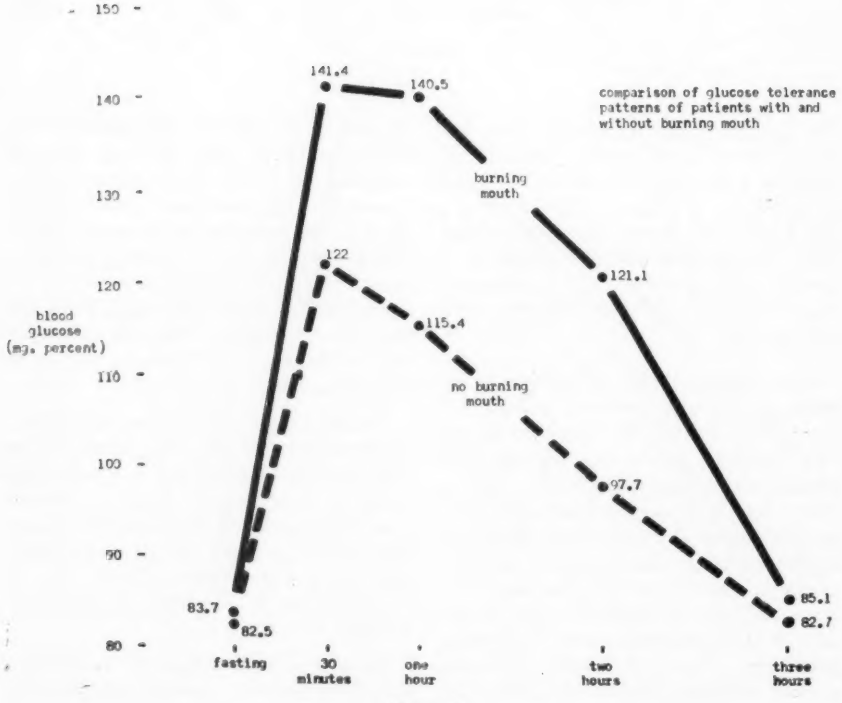

Figure 6

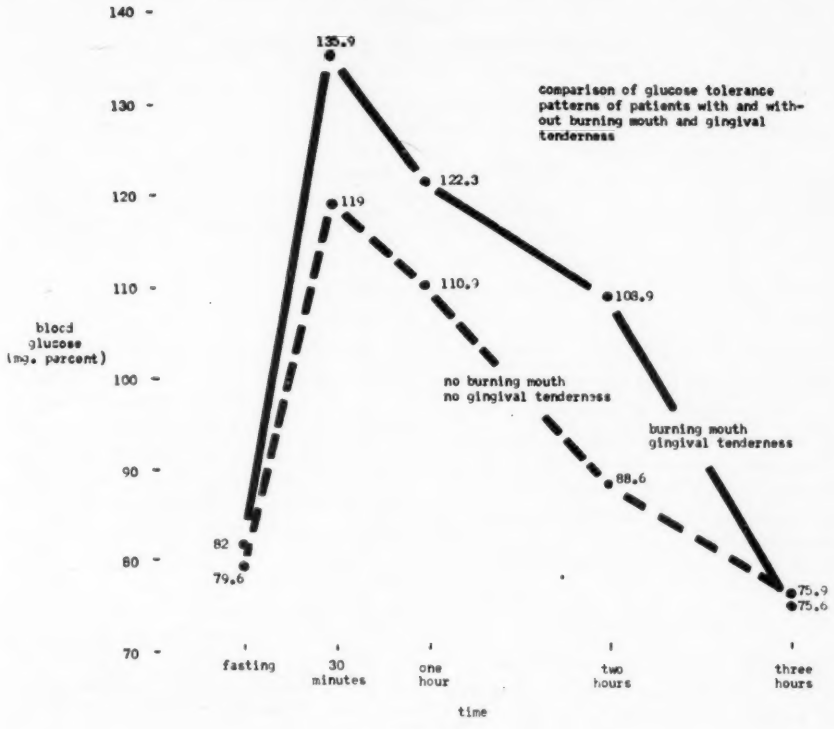

Figure 7

both of these symptoms. Figure 7 shows the glucose tolerance patterns of these two groups. Five points deserve special consideration. First, the symptomatic group demonstrates a relative decrease in glucose tolerance. Secondly, the smallest mean difference occurs at the three-hour level. Third, the greatest mean difference is observed at two hours. Fourth, the three-hour levels are lower than initially for both groups. Fifth and last, it is clear from Table 2 that there is no statistically significant difference in the glucose tolerance patterns of these two groups.

Dry Mouth and Gingival Tenderness: Sixty-six of the 100 patients complained with both of these symptoms or were asymptomatic with respect to these two items. Actually, 23 were asymptomatic and 42 symptomatic. Figure 8 shows the glucose tolerance patterns obtained from these two groups on the basis of the presence or absence of these complaints. Five points are deserving of mention. First, the symptomatic group shows a decreased tolerance to glucose. Secondly, the smallest mean differences occur at the fasting level. Thirdly, the greatest mean difference is noted at two hours. Fourthly, the three-hour level of those without symptoms is less than initially while the converse is true for those with dry mouth and gingival tenderness. Fifth and last, it is clear from Table 2 that there is no statistically significant difference between the glucose tolerance patterns of the two groups. Parenthetic mention should be made that the two and three hour mean differences come close to being significant ($P > .05$).

TABLE 6
Comparison of Burning Mouth and Age

Age groups	No burning mouth		Occasional burning mouth	
	Number of patients	Percentage of patients	Number of patients	Percentage of patients
0- 9	1	2		
10-19	9	10		
20-29	19	22		
30-39	24	27	3	22
40-49	16	19	3	22
50-59	14	16	4	28
60-69	3	4	4	28
Total number of patients	86		14	
Total percentage of patients		100		100

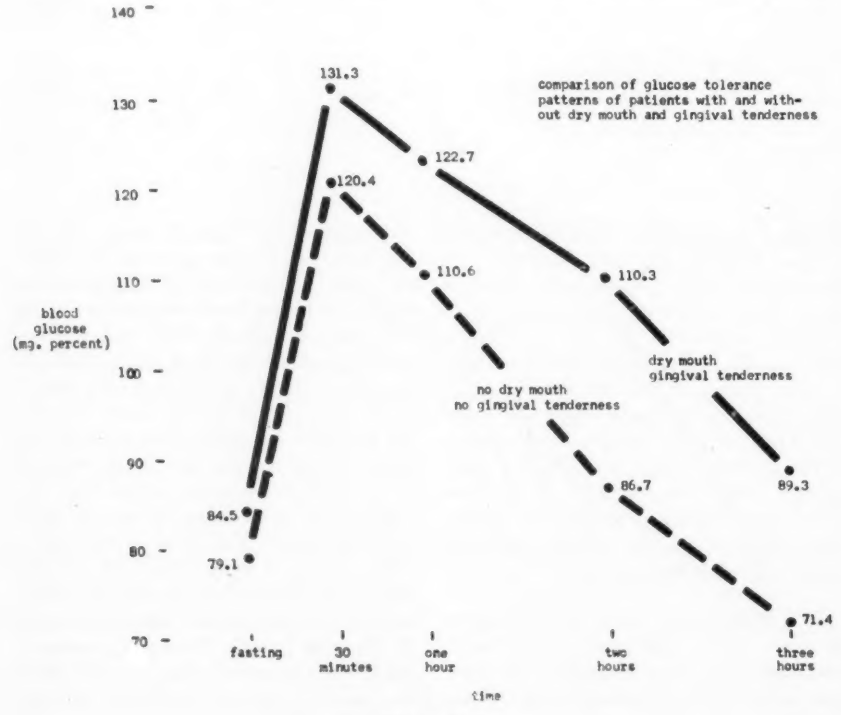

Figure 8

TABLE 7

Statistical Comparison of Mean Differences in the 40-49 Year Age Group

		Blood glucose differences				
		Fasting	*30 minutes*	*60 minutes*	*Two hours*	*Three hours*
Dry mouth-burning mouth versus no dry mouth-no burning mouth	Mean difference	14	52	77	56	19
	P	>.400	<.025	<.025	<.050	>.400

Dry and Burning Mouth: Eight patients reported both dry and burning mouth while 58 were asymptomatic in terms of these two items. Figure 9 shows the glucose tolerance patterns of these two groups. Five points warrant attention. In the first place, the symptomatic group shows relative decreased tolerance to glucose. Secondly, the smallest mean difference occurred in the fasting determinations. Thirdly, the greatest mean difference can be observed at the two-hour level. Fourth, the three-hour level of the asymptomatic group is lower than the fasting mean; the converse is true for those with symptoms. Fifth and last, there are significant mean differences at the thirty minute and one and two hour levels.

Combination of All Three Symptoms: Five patients reported the combination of dry and burning mouth and gingival tenderness. Thirty-eight were asymptomatic. Figure 10 shows the glucose tolerance patterns derived from these two groups. Five points are worthy of mention. First, the symptomatic group shows decreased glucose tolerance. Secondly, the smallest mean difference is observed at the fasting level. Thirdly, the greatest mean difference is recognizable at the two-hour interval. Fourthly, the three-hour blood glucose level of the asymptomatic group is less than initially; the three-hour level of the symptomatic patients is similar to that observed initially. Fifthly, the fasting, thirty-minute, and the two-hour mean differences are all statistically significant.

DISCUSSION

The data will be analyzed in three different ways: (1) a group analysis by comparison of the findings by Mosenthal and Barry with those observed in this study, (2) a subgroup analysis on the basis of symptomatology, and (3) a discussion of the age factor as it relates to blood glucose and oral symptoms.

GROUP ANALYSIS

It should be recalled that the present standards for the normal glucose tolerance curve include a fasting blood glucose less than 100 mg. percent, no blood glucose level greater than 150 mg. percent, and a return to below 100 mg. percent in two hours. On the basis of these criteria, two possibilities can be entertained. If all of the above specifications must be met for normal or abnormal glucose tolerance, then the dental patients in this survey cannot be regarded as systemically healthy. This conclusion follows from the fact that significant differences between the Alabama and Mosenthal and Barry groups were observed at the 2-hour period (Table 2 and Figure 3). If, on the other hand, normality and abnormality are to be judged on the basis of specific blood glucose criteria, then the dental patients must be regarded as either well or ill depending upon the specific yardstick employed. For example, on the basis of the fasting, 30 and 60 minute determinations, the dental patients meet the specifications for normality. However, utilizing the 2-hour determinations, the dental

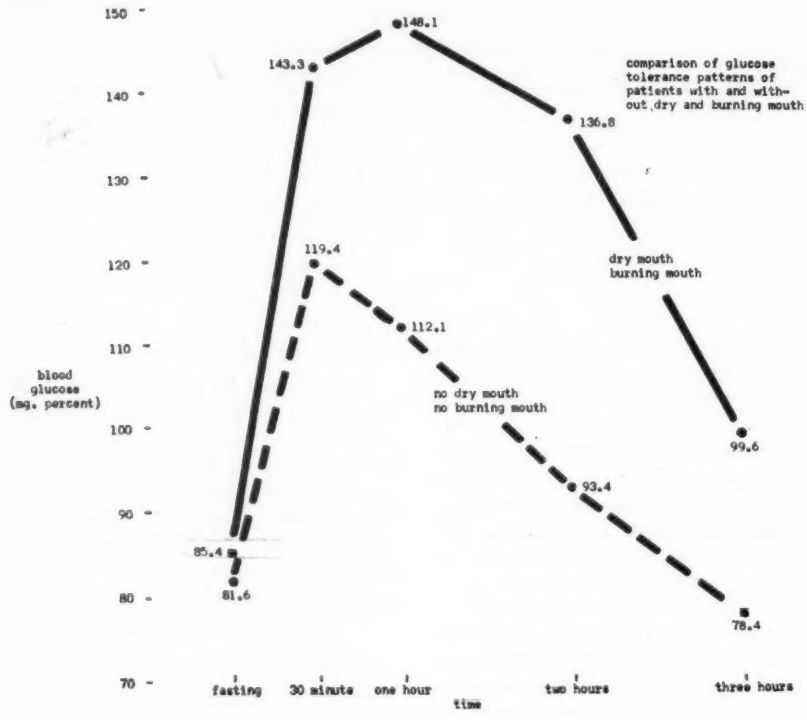

Figure 9

patients meet the requirements for systemic illness.

SUBGROUP ANALYSIS

It is noteworthy that, with regard to single complaints, only stomatopyrosis appears to delineate the diabetic from the normal patient (Figure 6). It should be recalled in this connection that significant differences between those with and without stomatopyrosis were observed at 30 and 60 minutes and at 2 hours (Table 2). Parenthetic mention should also be made that the 2-hour reading for those patients with stomatopyrosis also met the specifications for abnormality according to the Mosenthal and Barry recommendations. However, the 30 and 60 minute determinations for the burning mouth group, though significantly different from the asymptomatic group, were below 150 mg. percent. Hence, according to Mosenthal and Barry, these individuals should be regarded as within normal limits. There is no question but that it is hazardous to attach any true significance to the symptom of stomatopyrosis. Though it is difficult to state exactly how important this particular symptom is, it seems logical that it cannot be accepted as a physiologic finding.

The observations with combinations of two of the symptoms are also enlightening. One observes no significant differences between burning mouth and gingival tenderness versus no burning mouth and no gingival tenderness and dry mouth and gingival tenderness versus no dry mouth and no gingival tenderness. However, as in the case of stomatopyrosis alone, there is a

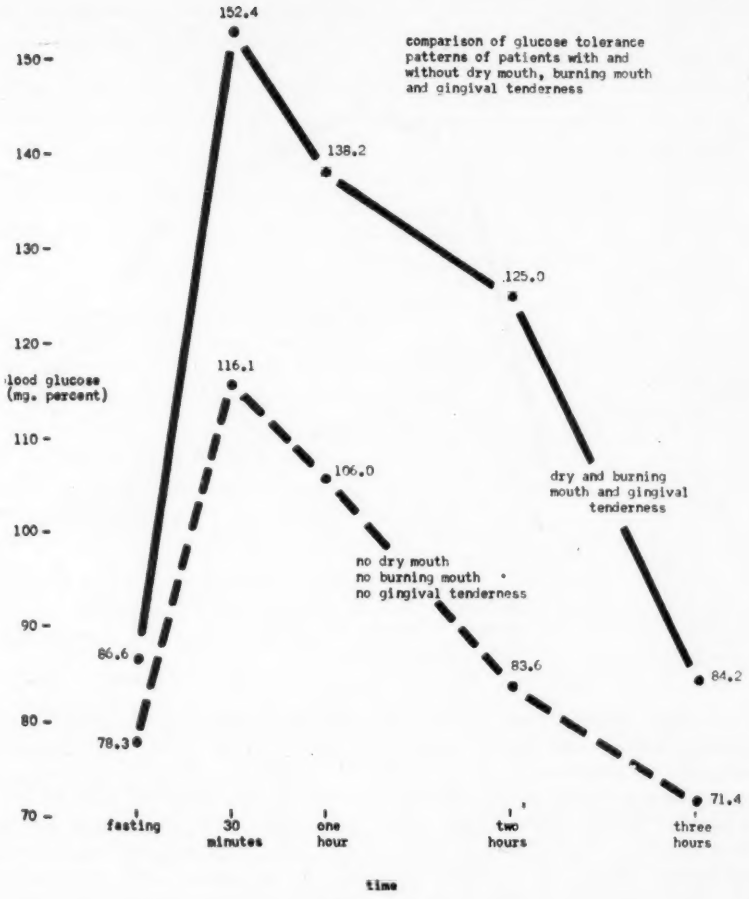

Figure 10

significant difference at the 30 and 60 minute and 2-hour determinations between those patients positive for stomatopyrosis and xerostomia versus those without these two symptoms (Table 2). The symptomatic group met the specifications by Mosenthal and Barry for abnormality at all points except in the fasting state. It can be observed (Figure 9 and Table 2) that the symptomatic group presented a fasting blood glucose below 100 mg. percent. Therefore, on the basis of fasting blood glucose, the patients with these two symptoms should be regarded as normal. Once again, it is dangerous to make any sweeping statements regarding the significance of these observations. However, it is difficult to believe that stomatopyrosis and xerostomia are representative of health.

The data derived from the combination of all three symptoms are particularly revealing. One can observe (Table 2) that the symptomatic and asymptomatic groups differ significantly in all determinations except the 3-hour sample. Also, most of the findings in the symptomatic group agree with the Mosenthal and Barry standards for abnormality. However, a statisti-

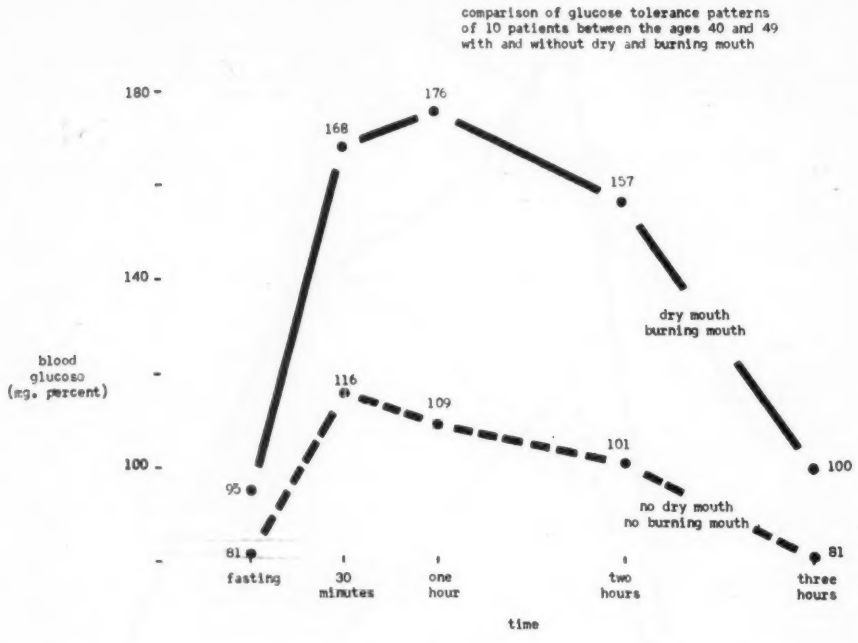

Figure 11

cally significant difference was observed in the fasting state between the two groups. Nonetheless, the symptomatic group still showed a mean value below 100 mg. percent. In other words, those patients with all three symptoms, on the basis of fasting measurements, would be regarded as healthy. Once again, it is difficult to imagine that the presence of these three symptoms is an index of well-being.

The most interesting observation is that the combination of symptoms produces more discrete curves than observed by an analysis of single symptoms. In other words, the symptomatic and asymptomatic groups become more clearly delineated as one employs combinations of symptoms rather than independent complaints. This is quite understandable since no disease process is represented by a single finding.

AGE FACTOR

There are some investigators who claim that blood glucose levels are higher in the older age groups. There is no disagreement that diabetes mellitus occurs more commonly in the autumnal years of life. Finally, it is clear from Tables 3, 4, 5, and 6 that the complaints of xerostomia and burning mouth are more often associated with the older age brackets. Therefore, the question which must be answered is whether the relationships which have been discussed in this report are true or whether they are complicated by the age factor. Accordingly, a restudy was made of those people with and without xerostomia and stomatopyrosis. However, the data were collected within one age group so as to eliminate the age factor. Figure 11 shows the glucose tolerance patterns for the symptomatic and asymptomatic patients in the 40 to 49 year age group. It is clear from Figure 11 that the two groups are clearly defined. More important, a comparison of Figure 11 with Figure 9 shows that at every temporal point the differences

are even greater for a specific age group than was observed for the entire 100 patients. Finally, a comparison of Table 7 and Table 2 shows that the age factor does not complicate the picture. These data suggest that the age factor is not a significant one in a consideration of the relationship between oral symptoms and blood glucose levels.

SUMMARY

1. On the basis of the overall glucose tolerance pattern, 100 routine dental patients appear normal if judged by the standards for glucose established by Mosenthal and Barry.

2. On the basis of the 2-hour determination in the glucose tolerance pattern, 100 routine dental patients would be regarded as systemically abnormal.

3. Burning mouth in contrast to xerostomia and gingival tenderness appears to be more related to diabetes mellitus.

4. The combination of stomatopyrosis and xerostomia seems to be more representative of the diabetic patient than any other combination of stomatopyrosis, xerostomia, and gingival tenderness.

5. The constellation of xerostomia, stomatopyrosis, and gingival tenderness appears to be more representative of the diabetic patient's complaints than any one or double combination of these three symptoms.

6. Within the limits of this study, the evidence suggests that the age factor does not play a role in the relationship of oral symptoms to the glucose tolerance pattern.

7. A report to follow[17] will analyze a number of *extraoral symptoms* (commonly associated with the diabetic state) and glucose metabolism.

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The Use of the Cornell Index to Determine the Correlation Between Bruxism and the Anxiety State: a Preliminary Report

by JACK L. THALLER, D.D.S.,* *Brooklyn, New York*

PERIODONTAL disease is the result of a multiplicity of factors, local and systemic. The disturbed psyche and the attendant chronic stresses can and frequently do alter the static composure of the body. That these factors definitely affect the homeostatic relationship has been established.[1,2,3]

Sorrin[4] has long viewed destructive oral habits as a contributing force in the causation of the periodontal syndrome. Campbell[5] states that emotional tension is expressed in various habits or bruxism. He further states that "bruxism is built up by the inability to express rage or hate." He continues "that the mechanical stress and strain were but a reflection or consequence of less tangible stress and strain within the personality." Bruxism, clenching, gritting of the dentition which are manifestations of the oral habit have contributed immeasurably to the destruction of the periodontium.[6]

PURPOSE

A pilot study was undertaken to determine if periodontal patients with a bruxism habit had a higher score within a personality inventory than patients without this habit; and to determine if a degree of correlation existed between bruxism and the emotional or anxiety state. Cameron[7] defines the anxiety state as "characterized by the presence of persistent heightened skeletal and visceral tension which disturbs a person's habitual rhythm of living and predisposes him generally, to give exaggerated and inappropriate responses to relatively slight provocation."

The study also investigated the possibility of a test which could be devised that would be used to help in diagnosis and treatment planning for the patient.

MATERIALS USED

The Cornell Index,[8] Form N2 is a series of questions referring to neuropsychiatric and psychosomatic symptoms which may serve as a standardized psychiatric history; and would statistically differentiate a person with serious personal and psychiatric disturbance from the rest of the population. The Cornell Index has shown itself to be effective in indicating the presence of the anxiety state, migraine, asocial trends and various other mental disorders. The score does not ascertain which specific difficulties are involved only that they are deviations from the average. It is the anxiety state with which we are concerned. The questions fall into two groups, those differentiating between persons with serious personality disturbances and persons without such disturbances. The reliability coefficient for one thousand subjects tested at five induction centers is .95%.[9]

METHOD

The Cornell Index was administered to 50 female patients at New York University, College of Dentistry, Department of Periodontia and Oral Medicine who had been diagnosed as having periodontal disease. Twenty-five of these were known to have the bruxistic habit which was determined by oral examination, visual observation and questioning of the patient and verified by the instructor or student present at the clinic chair. The patients were instructed in the purpose and method of answering the inventory. The only requirement was the ability to read and write. The inventories obtained were then submitted to a clinical psychologist for evaluation and interpretation.[10]

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RESULTS

The scores obtained for the two groups, the bruxistic and the non-bruxistic were tabulated by a clinical psychologist[11] are as follows.

Statistical analysis indicates there is a significant difference between the two groups. The difference between the two groups is significant statistically and is not accounted for by sampling errors.[13]

TABLE FOR COMPUTING THE MEAN AND STANDARD DEVIATION OF THE NON-BRUXISTIC GROUP

Scores	*Interval*	*Midpoint*	*Frequency*	*Class Marks in Terms of New Variable* X^1	X^1f	X^2f
0(2)	0-2	1	9	3	27	87
1(4)	3-5	4	6	2	12	24
2(3)	6-8	7	7	1	7	7
3	9-11	10	1	0	0	0
4(2)	12-14		0			
5(3)	15-17	13	1	−1	−1	1
6(2)	18-20	16	0	−2	0	0
7	21-23	19	1	−3	−3	9
8(4)	24-26	22	0	−4	0	0
10						
13		25	1	−5	−5	25
16			26		37	153
26						

TABLE FOR COMPUTING THE MEAN AND STANDARD DEVIATION OF THE BRUXISTIC GROUP

Scores	*Interval*	*Midpoint*	*Frequency*	*Class Marks in Terms of New Variable* X^1	X^1f	X^2f
2	0-4	2	2	3	6	18
4	5-9	7	9	2	18	36
5(4)	10-14	12	5	1	5	5
7(2)	15-19	17	4	0	0	0
8(3)	20-24	22	3	−1	− 3	3
10	25-29	27	6	−2	−12	24
12	30-34	32	1	−3	− 3	9
13	35-39	37	1	−4	− 4	16
14(2)			31		7	111
15(2)						
17						
19						
21						
22						
24						
26(2)						

CONCLUSION

1. On the basis of the results obtained in the preliminary survey a definite correlation exists between the anxiety state and the bruxistic habit.

2. The results show that periodontal patients with the bruxistic habit have a higher score on the Cornell Index than patients with non-bruxism.

SUMMARY

1. The Cornell Index was administered to 50 female patients at New York University College of Dentistry, Department of Periodontia and Oral Medicine, 25 with the bruxistic habit and 25 without the bruxistic habit.

2. A significant difference in the scores of the two groups on the Cornell Index was found to be 3.10.

3. The difference between the two groups is significant and can not be accounted for by sampling errors.

4. Further study is necessary.

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SOUTHERN ACADEMY OF PERIODONTOLOGY

The Southern Academy of Periodontology will hold its annual meeting on June 11th through 14th at the Ponte Vedra Inn in Ponte Vedra, Florida. Dr. Donald Kerr from the University of Michigan and Dr. Cliff Oschenbein of Austin, Texas will be the principal clinicians. Five limited attendance clinics will be given by Academy members. A truly great periodontic seminar has been timely planned with an abundance of recreational and social activities for all the family at picturesque Ponte Vedra. A.D.A. members are welcomed as guests.

For more information write Dr. J. J. Morris, Secretary, 101 Third Street, N. E., Atlanta, Georgia.

What Motivates Children to Practice Good Oral Hygiene?*

by NANCY J. DUDDING AND JOSEPH C. MUHLER, *Bloomington, Indiana*

IT IS generally agreed by most dental scientists that immediate brushing of the teeth following the ingestion of food is an effective means of reducing dental caries. Such an idea is part of the general concept that a clean mouth is associated with a decreased incidence of oral disease. It would seem important to learn why children do or do not brush their teeth, as well as what factors motivate them to have either good or poor oral hygiene.

EXPERIMENTAL

A total of 374 children, ages 6 to 14 years, all of whom resided in Bloomington, Indiana, were the subjects for this investigation. The examiner went into individual rooms of the school unannounced and estimated the level of oral hygiene in each child according to whether or not their teeth were clean, as evidenced by being free of materia alba and pigmentation. This was obviously a very subjective diagnosis; but, since considerable experience had been gained in conducting similar studies previously, it was rather easy to classify each child as having either "good" or "poor" oral hygiene. After the oral hygiene was determined on the individual child, he or she was asked only one question: "Where did you learn to brush your teeth?" No other comments or suggestions were made, and the child's answer was recorded on his examination sheet.

DATA AND DISCUSSION

The data obtained in this study are found in table 1. It is seen that of the 374 children, 81 (22%) were classified as having "good" oral hygiene and 293 as "poor." This figure for good oral hygiene is somewhat greater than that found in a previous survey (1) which indicated only about 6 per cent of the children practiced good oral hygiene. In any event, the number of subjects who practice good oral hygiene is not as great as those who have poor oral hygiene habits.

The data obtained from the children in response to the question: "Where did you learn to brush your teeth?" are found in table 1. There appears to be significant differences in the responses obtained from the children classified as having "good" or "poor" oral hygiene. The one which appears most significant is in relation to those responses concerning the effect of the dentist. Of the children classified as having "good" oral hygiene, 61% said they learned this oral hygiene practice from the dentist, while only 33% of those in the "poor" group said that the dentist was responsible for teaching them how to brush their teeth. If repeated surveys corroborate such findings as reported here, one would be able to provide real proof of the dentists' influence on the practice of good oral hygiene habits by children. Of equal impor-

TABLE 1

Distribution of Answers According to "Good" or "Poor" Oral Hygiene to the Question: "Where Did You Learn to Brush Your Teeth?" from 6 to 14 year old children.

Those children who were classified as "Good" (in per cent)	*From whom the children said they learned their brushing habits*	*Those children who were classified as "Poor" (in per cent)*
61	Dentist	33
4	Hygienists	11
19	Parents	4
2	Television	15
6	School	0
8	Did not know	37

*This study was supported in part by a grant from The Procter and Gamble Company, Cincinnati, Ohio.

tance is the clinical impression obtained from these examinations that when the dentist spends some time with the child in teaching him how and when to clean his teeth, the child will respond by having a clean mouth. If a cleaner mouth is truly associated with less oral disease then a good patient response should stimulate the dentist to continue (or begin) such practices with his patients in the attempt to reduce the burden of dental caries and periodontal disease in his practice and community.

The influence of the hygienist in teaching oral hygiene was not demonstrated in this survey. This may be explained on the basis that the hygienist does not routinely see young patients with any frequency except to give topical fluoride applications. It would seem highly desirable, however, to use such auxiliary dental personnel for dental education purposes in the office and thus further the general practitioner's role in preventive dentistry as has already been suggested (2).

The contribution of the parents to the children's practice of oral hygiene is expressed by noting that 19% of the children who were classified in the "good" group said that they had learned to brush their teeth from their parents. Of added interest, 80% of these parents said that they, in turn, had learned their oral hygiene habits from their family dentist. This is contrasted with only 4% of the children in the "poor" group who replied positively that their parents had taught them how to brush their teeth. When these parents were questioned as to where they learned how to brush their teeth, only 15% said they had such information given them by their dentist.

One of the interesting aspects of this study was the number of positive replies the children in the "poor" group gave stating that television was the medium through which they learned how to brush their teeth. Apparently this medium was not effective in promoting good oral hygiene in this group, at least under the conditions that it is now used.

The authors readily realize that such information as obtained in this study has severe limitations. Certainly these data should not be used as a basis of making similar conclusions as to the population as a whole. However, these data do seem to suggest that the dentist has a pronounced influence on his patient's desire to practice proper oral hygiene. The obvious extension of this idea is that the dentist should spend more time on dental education with his individual patients since better oral hygiene practices in the individual patient seem to result from his efforts. It seems only logical to suggest that the best and most effective means for patients to learn about dental health is from the dentist. The only apparent limiting factor in extending such practices to the entire population is the fact that too few people routinely see their family dentist.

CONCLUSION

A total of 374 children, ages 6 to 14 years, were examined for their oral hygiene and classified as "good" or "poor." In addition, each child was asked where he learned to brush his teeth. It was found that of those classified as having "good" oral hygiene, 61% said that they learned such practices from their dentist. In contrast, only 33% of those classified in the "poor" group provided such an answer. The influence of the dentist of promoting good oral hygiene in the individual patient is discussed.

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An Evaluation of the Reliability of Radiographs in the Measurement of Bone Loss in Periodontal Disease*

by JØRGEN THEILADE, L.D.S., B.SC.D.

THERE is need for reliable information concerning the epidemiology of periodontal disease. In order that data obtained by various surveys can be compared, similar methods of examination must be used. However, periodontal disease is an entity under which pathologic conditions, differing widely in appearance, are grouped, and there is a lack of uniform definition with regard to criteria characteristic of this disease. Therefore, indices to be used in epidemiologic surveys to express the degree of periodontal disease are often based on only one or a few of the generally accepted criteria of this disease.

One of the criteria, which may be measured in units, is the loss of supporting bone around the teeth as determined by the use of radiograms. This sign is therefore frequently used in comparative studies.

SURVEY OF THE LITERATURE

Belting[1] in a review of the epidemiology of periodontal disease classified the different indices into four groups on the basis of the methods used for determination of the score, namely: (1) examination of tooth loss; (2) radiographic examination alone; (3) clinical examination alone; and (4) correlation of clinical and radiographic examinations.

Three indices were found to be of value in a quantitative evaluation of bone loss in periodontal disease, namely those proposed by Miller and Seidler,[2] by Sheppard,[3] and by Russell.[4] Miller and Seidler, and Sheppard used indices which are based on radiographic examinations of some or all teeth. Their systems use radiographs only to determine the state of periodontal disease. Both systems use the degree of bone loss around the teeth as a measure of the severity of periodontal involvement. The Russell index is based on clinical findings with additional information from radiograms giving the degree of bone loss around the teeth. Russell stated that using clinical observations alone would underestimate the condition in three to ten percent of the cases. One feature of the Russell system is that it is a combination of an index for gingivitis and for periodontal disease. He also examined the errors in the use of his system due to disagreement between different examiners.

However, it may be incorrect to assume that a tooth which has lost its bone support along half the length of the root has also lost half its total area of attachment of root surface to alveolar bone. Goldman, Schluger and Fox[5] state that the resorption of bone along one third the length of the root may indicate that the tooth has lost two thirds its total surface for attachment to bone, dependent on the shape of the root. They further state that root investment must be thought of in square millimeters of attachment rather than in profile proportions.

Brown[6] measured the areas of loss of attachment due to bone recessions of two,

*From a study submitted in partial fulfilment of the requirements for the Bachelor of Science Degree in Dentistry at the Department of Periodontics and the Division of Postgraduate Studies, Faculty of Dentistry, University of Toronto, May 1958.

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four, or five millimeters and related these areas to the total root surface area. He found that this relation varied with the shape of the root. Whyte[7] reported a method for measuring the area of the root surface of extracted cuspid teeth. He reported that root surface area could not be accurately predicted from any single measurement. He estimated that predictions of total root area, calculated from the area of the radiographic silhouette, might be accurate within the range of plus or minus fourteen percent.

STATEMENT OF PROBLEM

This study was undertaken (1) to test the reliability of radiographs in the application of the indices proposed by Miller and Seidler, by Sheppard, and by Russell; (2) to compare the independent findings of four periodontists using the three indices mentioned above, and (3) to compare the relative amount of bone loss on the four main surfaces of the teeth examined.

Sheppard graded the degree of bone resorption from zero to ten. "The number 'one' signifies just enough loss to be visible on the . . . radiograph. . . . Thus 'five' indicates the loss of half the alveolar bone, while 'ten' implies complete loss of alveolar support. Intermediate numbers denote corresponding degrees of bone loss." He evaluated radiographically six regions of the dental arches and gave the average of these six region values as the index of the particular case.

Miller and Seidler used essentially the same system. The dental arches were divided into four segments. "The extreme amounts of periodontal experience for each of the four segments were recorded as 0 to 5. An index of 0 was ascribed to a segment when there was no bone dissolution evident; 5 units were given when the bone was destroyed to the apices of the teeth. An index of 3 was recorded when half the bone in a segment had disappeared. Units of 1, 2, and 4 were estimated as fractions of 3 and 5."

The Russell system for scoring and classification of periodontal disease differs from the above mentioned two systems in that it is based mainly on clinical examinations, although additional radiographic criteria are applied in the clinical test. The index produces a combined score for gingival disease and periodontal disease. The figures zero, one, two, four, six, or eight may be reported as the score for each tooth to describe the result of gingival or periodontal involvement. Numbers one and two designate different degrees of gingivitis. Radiographic examination does not apply in degrees one and two. Number four describes early periodontal disease seen in the radiograph as "notchlike resorption of the alveolar crest." There is no corresponding clinical criterion for this state. Figures six and eight designate various degrees of periodontal disease as determined by clinical examination plus radiographic examination.

MATERIAL AND METHOD

The material for this study was the product of dissections in the Anatomy Laboratory, University of Toronto. It consisted of dried human skeletal specimens comprising 39 pieces of mandibles in which teeth were present. The specimens exhibited gross evidence of the effects of periodontal disease. The jaws originated from 31 persons who died in Canada within the last 30 years. Their ages at death ranged from 21 to 84 years. The mandibles were all divided sagittally some place in the incisor region, and some of them were divided some place in the ramus of the mandible as well. As the incisors were always more or less damaged during the sectioning of the mandibles, these teeth were not examined in this study. The portions of mandibles which were radiographed enclosed between 20-25 each of cuspids, first and second bicuspids, and first, second, and third molars—a total of 133 teeth. As the ideal angulation between the film and the long axis of the teeth is not so easily obtainable in radiography of the upper teeth, no maxillary jaws were used in this study.

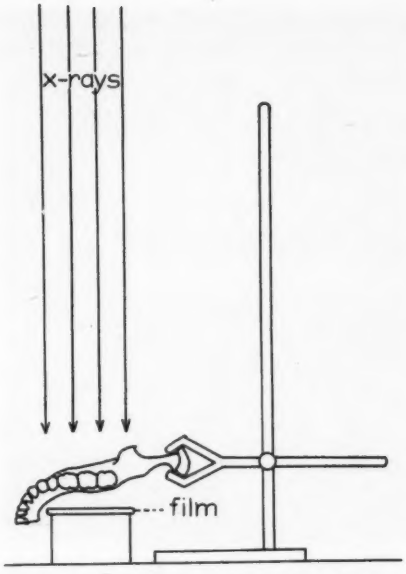

Fig. 1. Diagram showing the radiography technique.

Each specimen was radiographed under similar circumstances. The film was placed on a table and the portion of mandible was secured in a rigid position. An attempt was made to place the long axis of the teeth parallel to the film. The distance from the central fossae of the teeth to the film was approximately three-quarters of an inch (Fig. 1). The thickness and the form of the bone around the teeth made this the shortest distance at which the film could be placed parallel to the long axis of the tooth in every case without bending the film. In order to have this distance as short as possible in each case, the film was placed lingual to the dental arch in the molar region, whereas it was placed buccal to the arch in the bicuspid and the cuspid regions. Goldman, Millsap, and Brenman[8] could see no difference in density or in the trabecular pattern when radiographs were taken from the lingual side of the tooth rather than from the buccal side. To avoid distortion of the image of the tooth, a long distance technique was applied. The focus point of the X-ray tube was approximately 32 inches away from the film. The exposure time in all cases was four-and-a-half seconds at 20 milliamperes and 70 kilovolts.

All specimens were radiographed twice: (1) in their original condition, and (2) with fine wires adapted to the alveolar edges around the teeth and to the base of the intrabony deformities. Along the lingual side of the alveolar crest a single wire of the dimension 0.01 inch was used, and along the buccal side two twisted wires of the dimension 0.007 inch was applied.

The radiograms of the same area with and without wires were designated with different numbers by a code known only to the investigator. They were submitted separately to the four periodontists who scored each tooth on the basis of the three indices which were employed. The four periodontists worked independently of each other.

For the teeth radiographed without wires, the score for each tooth was computed by averaging the scores for the mesial and the distal surfaces. For the teeth with wires indicating the position of the alveolar crest around the circumference of the root, the score for each tooth was computed as the average for the mesial, distal, buccal, and lingual surfaces.

This method of study produced a scoring for each tooth (a) with and without wires on the alveolar edge; and (b) by four different periodontists and by (c) three different systems.

FINDINGS

Since scorings from radiographs with wires present were probably more accurate than those without wires, the scoring achieved from films with wires was used as the standard with which to compare the scores registered from films without wires. This report will therefore indicate the difference in findings from films without wires as underestimation or overestimation of bone loss as related to the scores from films with wires.

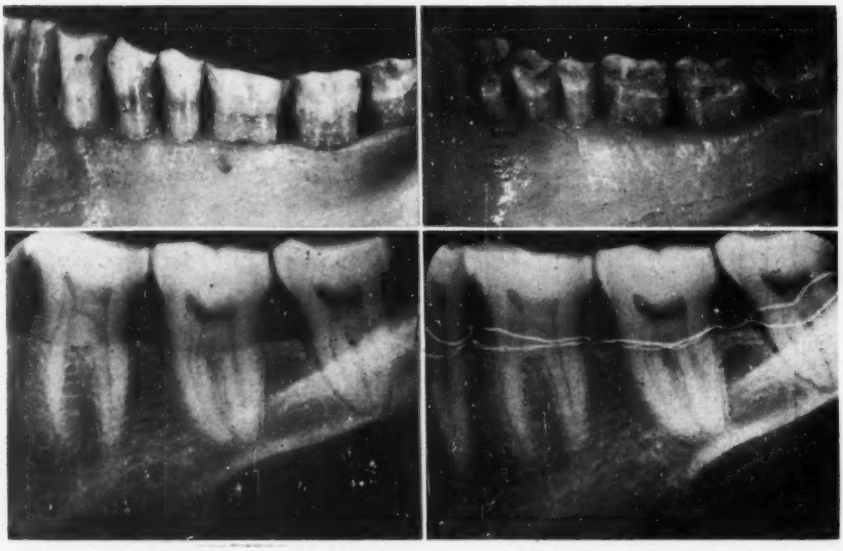

Fig. 2. Above, photographs of jaw No. 117R, buccal and lingual views. The photograph of the buccal has been turned, in order to have mesial surfaces to the same side for both photographs and radiographs. Below, left, radiograph of same specimen. On right, radiograph taken after two twisted wires were adapted along the buccal bone edge and a single wire along the lingual bone edge.

In many cases there was good agreement between the two average scores given each tooth by any of the periodontists according to any one of the three different indices. The differences were considered to be of minor significance when they were numerically less than one unit in the Sheppard system or 0.5 unit in the Russell system and the Miller and Seidler system.

A significant underestimation of the bone loss was said to have occurred if the average score for a tooth evaluated from a radiogram without wires was one unit or more lower according to the Sheppard system than the corresponding average score determined from the radiograph with wires (or 0.5 unit or more for the Russell system and the Miller and Seidler system). The percentage of teeth for which an underestimation of bone loss occurred varied greatly depending on the type of tooth, the examiner, and especially on the index used. When the Russell system was used, between 17 and 71 percent of the different teeth were scored too low when the wires were not present. The corresponding range for the Sheppard system was between 4 and 52 percent, and for the Miller and Seidler system, between 5 and 52 percent.

A significant overestimation of bone loss was said to have occurred if the average for a tooth without wires, according to the Sheppard system, was one unit or more greater than the corresponding average score determined from the radiogram with wires. (Again the borderline for the Russell system and the Miller and Seidler system was 0.5 unit.) It was found that an overestimation of bone loss did not occur in nearly as many cases as an underestimation. The percentage of teeth for which an overestimation occurred using the Russell system varied between 0 and 35 percent. The corresponding range for the Sheppard system was between 0 and 15 percent, and for the Miller and Seidler system it was between 0 and 25 percent.

Generally, it can be said that the percentage in which there was an underesti-

Fig. 3. Photographs and radiographs of jaw No. 147R. The radiograph with wires shows a marked and irregular loss of alveolar bone on the buccal surfaces of the bicuspids and the first molar. Thinned interdental alveolar tips are seen.

mation of bone loss in radiographs without wires was lower for the Sheppard and the Miller and Seidler systems than for the Russell system. The Sheppard system and the Miller and Seidler system seem to be of similar accuracy in estimation of bone loss.

In order to determine whether the disagreement between the two average scores given to each tooth, using any one of the three systems, originated from differences in the scores with and without wires for one or both of the approximal surfaces, or whether it originated from the additional information of bone loss on the buccal and the lingual surfaces after the wires were adapted, separate comparisons of the scores with and without wires were made for the mesial and distal surfaces. These comparisons revealed in most cases good agreement between the scores with and without wires for one or both of the approximal surfaces. The disagreement between the two average scores was, therefore, most frequently due to the fact that more bone loss was detected when the wires were present on the buccal and lingual surfaces. Illustrations hereof are given in Fig. 2 and Fig. 3.

Fig. 2 shows the photographs of jaw No. 117R and the corresponding radiographs. Examiners 1, 2, and 3 gave the same mesial and distal scores for the second molar, using the radiographs with and without wires and all three scoring systems. There were, thus, no differences between the scores with and without the wires for these two surfaces. This was the most frequent finding when the scores for the mesial or the distal surfaces with and without wires were compared.

The first molar in Fig. 3, showing photographs and radiographs of jaw No. 147R, was given the same scores by examiner 1 for the mesial and the distal surfaces as judged from the two radiographs according to the Sheppard system. However, the additional information for the buccal and lingual surfaces obtained from the radiograph with wires raised the average score 1.75 units. The bone loss was underestimated when the radiogram without wires was examined.

TABLE I

Average units of bone loss on the different surfaces of each type of tooth as determined from the radiographs with wires by examiners 1, 2, 3, and 4, according to the Sheppard system. In the column "avr." the four examiners' scores for the same surface are averaged, giving an average score for each surface of each type of tooth

Type of tooth	No. of teeth	Average score for all mesial surfaces					Average score for all distal surfaces					Average score for all buccal surfaces					Average score for all lingual surfaces				
		ex. 1	ex. 2	ex. 3	ex. 4	avr.	ex. 1	ex. 2	ex. 3	ex. 4	avr.	ex. 1	ex. 2	ex. 3	ex. 4	avr.	ex. 1	ex. 2	ex. 3	ex. 4	avr.
cuspids	20	1.7	1.8	2.6	2.4	2.1	1.3	1.7	2.3	2.2	1.8	3.2	3.7	4.1	3.8	3.7	1.4	1.8	2.7	2.6	2.1
1st bicuspids	24	1.3	1.8	2.3	2.0	1.8	1.5	2.0	2.3	2.5	2.1	3.3	4.0	4.0	3.3	3.6	1.1	2.0	2.3	2.2	1.9
2nd bicuspids	23	1.4	2.1	2.1	2.0	1.9	1.6	2.5	2.4	2.7	2.3	2.3	3.1	3.0	3.3	2.9	1.4	2.1	2.4	2.5	2.0
1st molars	21	1.3	1.7	1.9	2.0	1.7	1.6	2.3	2.1	2.5	2.1	2.9	3.3	3.2	3.2	3.1	1.5	2.5	2.2	2.4	2.1
2nd molars	25	1.7	2.2	2.2	2.4	2.1	1.7	2.2	2.1	2.5	2.1	2.6	3.4	3.1	3.1	3.0	1.6	2.6	2.5	2.5	2.3
3rd molars	20	2.1	2.5	2.5	2.9	2.5	1.4	1.8	2.2	2.3	1.9	1.2	2.1	2.0	2.3	1.9	1.9	2.6	2.2	2.6	2.3

In Table I are indicated the mean scores for all mesial, distal, buccal, and lingual surfaces of each type of tooth as scored by the different examiners with wires indicating the bone levels in the radiographs. For simplicity, only the values obtained from Sheppard's system are given in this table. It is seen that although the examiners tended to score similarly to each other, examiner 1 scored invariably lower than examiners 2, 3, and 4.

In the table the four examiners' scores for the same surface are averaged, giving an average score for each surface of each type of tooth. These average scores range for mesial surfaces from 1.7 to 2.5 units; for distal surfaces, from 1.8 to 2.3 units; for lingual surfaces, from 1.9 to 2.3 units; and for buccal surfaces, from 1.9 to 3.7 units. The wide range for the buccal surfaces is due to the value of 1.9 units for buccal surfaces of the third molars. All the other values for buccal surfaces range from 2.9 to 3.7 units. The table thus indicates that for all types of teeth examined, except for the third molars, the greatest bone loss was on the buccal surface. For the third molar, the greatest amount of bone loss was found on the mesial surface, and the next greatest loss was on the lingual surface.

DISCUSSION

The findings in this study reveal that a severe underestimation of bone loss, due to the lack of information of bone level on the buccal and the lingual side of the tooth in an ordinary radiogram, may occur in a considerable percentage of the teeth examined.

The probability of this error in the evaluation of radiographs has been recognized by previous investigators. Sheppard[8] states that buccal and lingual losses are not visible in radiographs while Day and Shourie,[9] using the Sheppard system in a survey of periodontal disease, state that frequently it is impossible to obtain a satisfactory view of the labial, buccal, and lingual bone loss in a radiogram, and that the score, therefore, is an estimate of the condition. Schei et al.[10] also realized this problem, and they state that their figures cannot be claimed to be absolute values, as the bone resorption is usually most advanced on the lingual and approximal surfaces. Goldman, Millsap, and Brenman[8] showed that the removal of the buccal and lingual bony plate had no effect on the radiograph except that there is a decrease in radiodensity.

Probably the best way to prevent the above mentioned misinterpretation is to

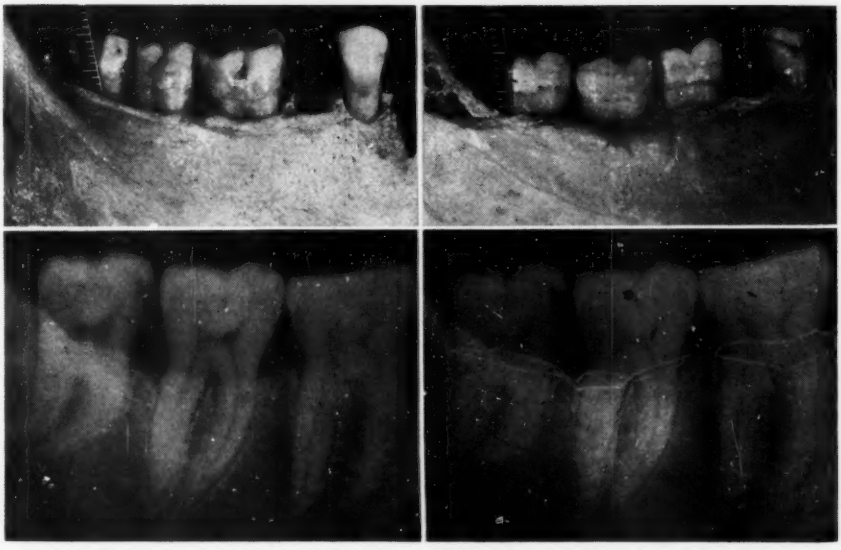

Fig. 4. Photographs and radiographs of jaw No. 164L showing an intrabony crater between the second and third molars. This was not detected from the radiograph without wires.

combine an evaluation of the radiogram with a direct clinical examination, as applied in the Russell index and in surveys carried out by Belting, Massler, and Schour[11] and Ramfjord.[12,13] The direct examination may detect recession of the gingival tissues or pocket formation at places where there is no detectable radiographic evidence of bone loss.

From Table I it is seen that all types of lower teeth examined in this study, except the third molars, had the greatest bone loss on the buccal side and that the lingual bone loss was of approximately the same magnitude as the mesial and distal. The buccal bony plate of the mandible is frequently thin, and this condition apparently predisposes to resorption of bone. For the third molar, the surface with most bone loss was the mesial, and the next most involved surface was the lingual. The relative lingual position of the third molar within the mandible frequently results in a thin lingual bony plate over the root of this tooth. On the buccal side of the third molar there is the external oblique ridge. This ridge is not likely to be resorbed, and if resorption does take place in close proximity to the tooth, it will apparently produce resorption of the vertical and intrabony pattern. Fig. 4 shows this situation. A probe, calibrated in millimeters, has been placed in the intrabony deformity.

The difference between the examiners' judgments is a problem in epidemiologic surveys. If different surveys are to be compared, many errors may be avoided by employing the same examiner through all studies. However, an index with well defined criteria will allow results from different examiners to be safely compared.

To compare the reliability of the three systems used in this study is difficult. The units in the systems of Sheppard and of Miller and Seidler are used entirely to determine the length of the apical migration of the alveolar bone. In the Russell system, the scores indicate the findings of direct clinical examination as well as amount of bone loss. The units by which bone loss is measured vary in the three systems. It is seen that loss of half the bone for a tooth,

according to the Russell system, is denoted as 75 percent of the highest number possible (six of eight); whereas the same amount of bone loss, according to the Sheppard system, is 50 percent (five of ten); and in the Miller and Seidler system, 60 percent (three of five). The choice of an index for a periodontal survey will therefore be dependent on the investigator's concept of the severity of the disease in relation to the amount of bone loss.

In this study, the variation in scores for the same tooth when scored by different trained periodontists is obvious. This is probably due to the fact that the distance to be measured in units is poorly defined. The cemento-enamel junction is used as a landmark from which the bone loss is measured. Ritchey and Orban[14] stated that the normal level of the interdental crest is slightly below a line between the cemento-enamel junctions of the two adjacent teeth. Belting, Massler, and Schour[11] considered bone loss to have taken place when the alveolar crest was found two millimeters or more from the cemento-enamel junction, while Schei et al.[10] registered bone loss when the alveolar crest was found more than one millimeter from the cemento-enamel junction. However, the curved cemento-enamel junction on the approximal surface is not clearly defined in the radiograph (see Fig. 5). In teeth with dental caries, or fillings involving the cemento-enamel junction approximally, even this landmark disappears from the radiogram. In a radiogram, the cemento-enamel junction is usually not visible on the buccal and on the lingual surfaces. Even when the level of the alveolar crest is easily seen on these two surfaces after the application of the wires, the distance to the cemento-enamel junction is still not easily determined.

There is also argument whether the distance between the cemento-enamel junction and the bony edge is normally constant all through life[15] or whether an increase of this distance with age is to be called physiologic.[16] However, if this migration is believed to occur, the age of the person should be taken into consideration when the score for the bone loss due to periodontal disease is given.

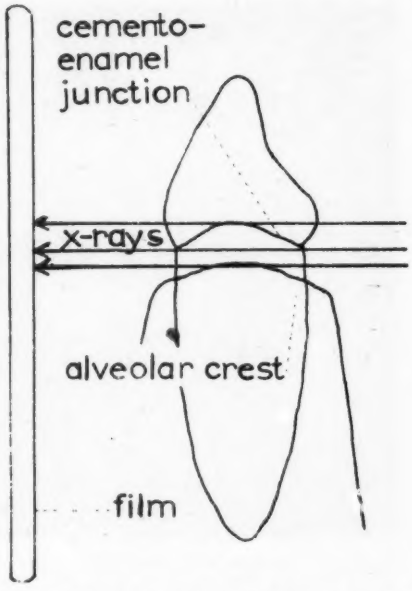

Fig. 5. Diagram showing a tooth with a curved cemento-enamel junction approximally. It is difficult to predict where on the film the image of the cemento-enamel junction will occur.

An interesting observation was often made from the specimens used in this study. Where the buccal bony plate was thin, a buccal recession seemed more likely, as shown in Fig. 6. Before determining the score to indicate bone loss around these teeth, one might ask the question whether this buccal bone loss is due to an apical migration of the bone edge as a result of disease or whether the bone has always failed to cover the buccal surfaces of the roots of the teeth to this extent, because of inadequate development originally or because the teeth erupted partially outside the bone.

In many cases there are various levels for the bone edge on a single surface of a tooth. If the scoring is based on the most apical point of bone loss, this will not con-

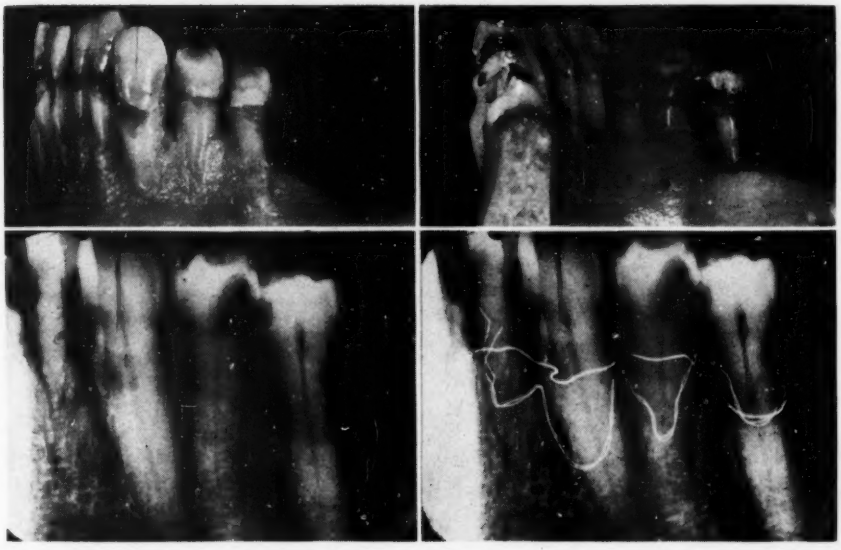

Fig. 6. Photographs and radiographs of jaw No. 138R. The photograph of the buccal view shows that the bone only partially covers the roots of the teeth. The radiograph with wires reveals the partial lack of bone on the buccal surfaces of the teeth. This radiograph also shows that the tip of the interdental crest will not be detectable if it is radiolucent as seen between the bicuspids.

vey an accurate picture of the total bone loss for that surface. See Fig. 3 and Fig. 7.

In some cases a greater score was given for an approximal surface after the wires were adapted to indicate the height of the crest. An example of this is shown in Fig. 4, where the radiogram indicates a higher bone level interproximally between the second and third molars than was actually present, as seen in the radiograph with wires. The explanation for this is given by the corresponding photograph which shows an interproximal bony crater not detectable in the radiogram, because of the projection on the film of the intact buccal and lingual bony plates.

In other cases, a lower score, indicating less bone loss, was given after the teeth were radiographed with wires. Fig. 3 and Fig. 6 show that a radiogram may be interpreted as showing a bone loss which is not actually present. The radiograph, without wires, of the second bicuspid in Fig. 3 was interpreted by all examiners as showing a greater bone loss for the mesial surface than for the distal surface. The photographs show that the bone level is the same on the mesial and the distal surfaces. The reason for the misinterpretation of the radiograph without wires is probably that the X-rays passed through the alveolar crest on the mesial side of the second bicuspid without giving a distinct shadow on the film, because the alveolar crest was thin and radiolucent. The same feature can be observed in Fig. 6 for the mesial surface for the second bicuspid.

Finally it should be mentioned that slight inaccuracy in the scores from radiographs with wires may be the result of the difficulties of bending and adapting the wires around the teeth at the edge of the alveolar bone. The wire for the buccal side may not have met with the wire from the lingual side exactly at the tip of the interproximal alveolar crest. The preservation varnish, with which the specimens were covered post mortem, or improper defleshment, may in some instances have pre-

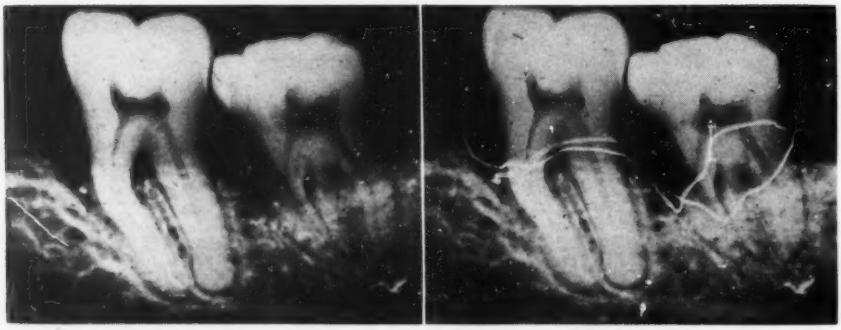

Fig. 7. Radiographs, with and without wires, of jaw No. 183R. The radiograph with wires shows that the bone edge buccally and lingually may be situated either coronally to or apically to the bone as shown at the mesial and distal surfaces.

vented the wires from coming in immediate contact with the bone tissue.

SUMMARY

A study has been carried out (1) to test the reliability of radiographs in the application of three different systems for scoring that have been employed to express the amount of bone loss in periodontal disease; (2) to compare the independent findings of four periodontists using the three indices mentioned above; and (3) to compare the relative amounts of bone loss on the four main surfaces of the teeth examined.

The material consisted of dried human skeletal specimens comprising 39 pieces of mandibles from 31 persons who died in Canada within the last thirty years. Their ages at death ranged from 21 to 84 years of age. The specimens exhibited gross evidence of the effects of periodontal disease.

The lack of a buccal and lingual view of the alveolar bone in the dental radiograph caused a serious underestimation of bone loss, due to periodontal disease, in a considerable percentage of 133 mandibular teeth examined, varying for the different types of teeth, the examiner, and the score system used.

The uniformity in judgment of the radiographs by four trained periodontists, was examined. Although their interpretation of radiographs varied, each single examiner seemed consistent in his judgment.

For mandibular cuspids, bicuspids, and first and second molars the greatest bone loss was on the buccal side. For third molars the greatest bone loss was on the mesial surface, and the next greatest bone loss was on the lingual side of the tooth.

ACKNOWLEDGMENTS

The many radiographs were examined by the following members of the staff of the Faculty of Dentistry, University of Toronto: C. H. M. Williams, D.D.S., B.Sc.D., Professor, Head of the Department of Periodontology; W. G. McIntosh, D.D.S., M.Sc.D., F.A.C.D., Associate Professor, Department of Periodontology; J. E. Speck, D.D.S., Associate Professor, Department of Periodontology; and D. L. Anderson, D.D.S., M.Sc.D., Research Associate in Dental Research, Division of Dental Research.

Valuable guidance during the performance of the investigation was rendered by R. M. Grainger, D.D.S., D.D.P.H., Consultant in Dental Research, Division of Medical Statistics, Ontario Department of Health; K. J. Paynter, D.D.S., Ph.D., Chairman, Division of Postgraduate Studies; and Dr. C. H. M. Williams, Faculty of Dentistry, University of Toronto.

J. E. Anderson, M.D., Lecturer in Anatomy, Faculty of Medicine, University of Toronto, placed at disposal the skull material which was used in this study.

Mr. John Philpot, Senior Technician, Visual Aids, Faculty of Dentistry, prepared the illustrations.

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AMERICAN ACADEMY OF PERIODONTOLOGY SPRING CONFERENCE APRIL 25 TO 27, 1960

The annual Spring Conference of the American Academy of Periodontology will be held at the Kellogg Foundation Institute in Ann Arbor, Michigan from Monday, April 25 through Wednesday, April 27, 1960.

The program will include discussions by Edward L. Quinn, M.D., William L. Morgan, Jr., John W. Keyes, M.D., Robert E. Burns, all of Henry Ford Hospital, Helmut A. Zander of Eastman Dental Dispensary, and Sigurd P. Ramfjord of the University of Michigan School of Dentistry.

Enrollment is limited to members of the American Academy of Periodontology and their guests. Room reservations may be made by writing to the Michigan Union, 530 South State Street, Ann Arbor, Michigan.

For further information write Dr. William R. Mann, W. K. Kellogg Foundation Institute, the University of Michigan, Ann Arbor, Michigan.

Histochemical Study of Sulfhydryl (Cysteine) and Disulfide (Cystine) Groups in Human Gingiva

by EDUARDO KATCHBURIAN, S. *Paulo, Brazil*

THE problem of the presence of a genuine horny layer in the human gingival epithelium, has been a subject of study of several authors. The question, however, has not been definitely explained.

Weinmann[1] presents a general review of the literature in which authors mentioned give diverse opinions regarding the presence or absence of keratin in human gingival epithelium. Several authors[2,3,4,5,6,7,8,9,10] conclude that keratin does exist in the gingival mucosa. However, the results obtained by Cabrini *et al*[11] and Trott[12] showed the possibility of keratinization, parakeratinization and even absence of keratinization in normal gingival epithelium. More recently, Turesky *et al*[13] admit also the presence of keratinized and parakeratinized cells. It should be pointed out that the above mentioned authors with exception of Turesky and col.,[13] utilized simple methods of staining for the identification of keratin.

According to Pearse,[14] in the process of keratinization aminoacids of the cystine (disulfide) and cysteine (sulfhydryl) type are involved. Consequently a histochemical study of these substances should indicate the presence or absence of keratin in an epithelial layer. This being the case, by using the Chèvremont and Fréderic reaction for sulhydryl and Pearse's method for disulfide groups, we attempted a histochemical study on the question of keratinization of the human gingival epithelium.

Department of Histology, Escola Paulista de Medicina.

*The material was obtained at the Santa Casa de Misericordia de São Paulo (Prof. Mario Graziani).

**This material was obtained at the Department of Pathology of the Maternidade de São Paulo (Dr. Antonio Luisi).

MATERIAL

In this study, forty specimens of human gingival mucosa were used, of which thirty-two* were obtained by biopsy of the anterior and posterior zones, both from the maxilla and the mandible with tooth extraction, and eight** from the gingiva of the human foetus.

The thirty-two specimens were obtained from patients whose ages varied from eighteen to seventy-eight years of age; nineteen were female and thirteen male.

The fragments of gingiva of approximately five millimeters were immediately immersed in 10 per cent formalin.

For comparison purposes the keratinized oral mucosa of the rat (fixed in 10 per cent formalin) was used as a standard. Both the human and rat material remained in the fixative for a period which did not exceed 24 hours. After fixation, the fragments were imbedded in paraffin; sections of 7 micra were obtained.

METHODS

The methods used were as follows:

1. Mallory's trichromic method for verification of the existence of the so-called horny layer (stains in orange color), and later comparison with the histochemical methods.

2. Chèvremont and Fréderic method for sulfhydryl groups (cysteine). According to Pearse[14] it is indicative of the presence of keratin. The Chèvremont and Fréderic method, when positive shows an intense blue color. For the verification of the specificity of the method, sulfhydryl groups were blocked with mercury bichloride, according to Lison[15] and Adams.[16]

3. Pearse's method for disulfide groups

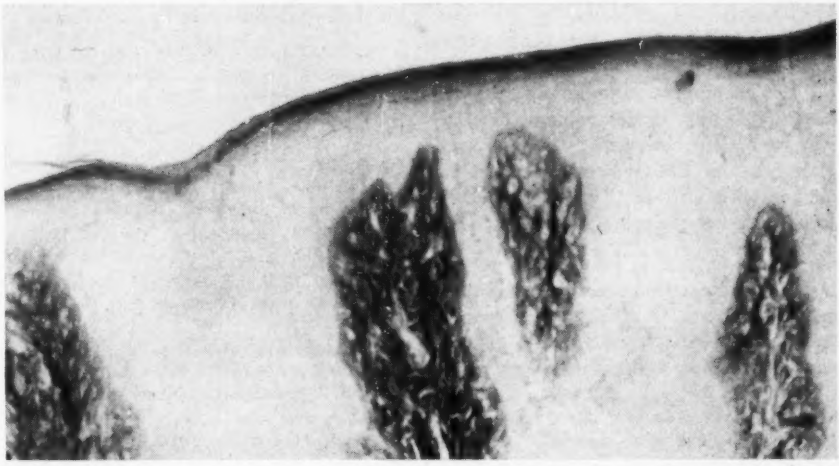

Fig. 1. Human gingiva. Observe the "Mallory positive" reaction in the entire epithelial surface. 500X.

(cystine). This method, established by Pearse,[14] shows an intense blue color, when positive.

RESULTS

Mallory's trichromic method. With this method, the majority of the sections of the human gingival epithelium revealed the presence of a sharp orange colored band, on the surface of the epithelium (fig. 1).

A minority presented orange colored zones distributed irregularly throughout the epithelium, and several sections presented only small spots of orange color on the more superficial zones.

As regards the rat, one could observe a thin and long orange band in entire epithelial surface.

Chèvremont and Fréderic method. With this method the reaction was negative.

The rat gingiva, however presents a clear and intense blue coloration, in the entire epithelium surface, thus indicating the presence of free sulfhydryl groups.

It should be stated that sections of the rat gingiva after being treated by mercury bichloride (blockade) showed a negative reaction to the Chèvremont and Fréderic method.

Pearse's method. The epithelium of the human gingiva and of the rat, were negative to method, since there was an absence of disulfide groups (cystine).

DISCUSSION

From the results obtained one concludes that keratin is absent at the level of the human gingival epithelium, in agreement with the findings of Turesky *et al*,[13] who demonstrated the presence of sulfhydryl and disulfide groups in the human gingiva.

The results, however, confirm in part those obtained by Cabrini *et al*[11] and Trott[12] since these authors also admit the possibility of non-keratinized gingival epithelium.

It was not possible to establish a relation between the presence of the orange color on the human gingival epithelium, obtained with the Mallory's method, and the blue color (Prussian blue) obtained in the same epithelium with the Chèvremont and Fréderic method, for in Mallory positive zones we find a Chèvremont and Fréderic negative reaction.

Although the majority of the authors[1,2,3,4,5,6,7,8,9,10] have made references to the presence of keratinized cells in the human gingiva, through simple processes of staining, in our opinion, the lack of correlation observed by us between Mallory's and Chèvremont and Fréderic methods demonstrate that we are unable to identify keratin using staining methods only.

We believe that only histochemical methods for the identification of keratin are feasible.

SUMMARY

Using histochemical reactions for sulfhydryl and disulfide groups the problem of the keratinization of the human gingival epithelium was studied.

Material from 32 biopsies and 8 necropsies of human gingiva was treated by Mallary's trichromic and Chèvremont and Fréderic methods for sulfhydryl groups and by Pearse's method for disulfide groups.

The keratinized mucosa of the rat was used as a standard. Negative reaction for sulfhydryl and disulfide groups were observed in human gingival epithelium; this was interpreted as indicative of the absence of keratin. It was not possible, however, to correlate the results obtained with Mallory's method with those of Chèvremont and Fréderic as in "Mallory positive" zones, negative Chèvremont and Fréderic reaction were observed. This fact shows that keratin cannot be identified with the use of staining methods only. The author concludes, therefore, that only with histochemical methods the identification of keratin would be feasible.

CONCLUSIONS

1. The staining methods did not indicate the presence or absence of keratin.

2. Histochemical methods are indicated for the identification of keratin.

3. In the material obtained, the human gingival epithelium was not keratinized.

The author wishes to thank Prof. Nylceo Marques de Castro and Dr. Wilson da Silva Sasso for their advice in the preparation of this study.

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Case Reports . . . .

A Case of Extreme Multiple Root Resorption with No Increased Mobility of the Teeth

by FRANK G. EVERETT, M.S., D.M.D., M.D.[1] AND FRANCIS J. FABRIZIO, D.D.S.[2]

WE HAVE had occasion to observe during the last six years an interesting case of extreme multiple root resorption. The patient, R. W. G., a white male born in 1923 presented to one of us (F. G. E.) on October 16, 1953, for treatment of a mild chronic marginal gingivitis. Full mouth roentgenograms taken at this time (Fig. 1) showed general root resorption, most extensively present in the region of all four upper incisors (Fig. 2). Involved next in severity were all four first molars. The lower incisors were also greatly shortened. All other teeth showed some signs of root resorption. There was no unusual mobility present. All teeth reacted vital to the electric pulp tester with the exception of the upper right central incisor; this tooth had been devitalized in an accident which the patient had suffered at the age of nine years. Extensive gingival recession was present in a number of teeth. The patient had undergone in his youth prolonged orthodontic therapy. Four different orthodontists had treated him at different times during adolescence and early adulthood. The first period of active orthodontic treatment had been from age 8 to age 9½, the second from age 11 to age 13½, the third one from age 16 to age 18, and the fourth one from age 20 to age 21.

The patient had suffered an attack of poliomyelitis at the age of 20 years. He had run a high temperature and had had muscular stiffness for the first week. He had been kept in bed for one month and then had spent two additional months convalescing. The disease left him with a slight limp in his right leg.

The history of the patient in regard to other past illnesses is negative with the exception of an allergic condition, hay fever. A recent thorough physical check-up yielded no significant findings. Results of hematologic alkaline Phosphatase, Calcium and Phosphorus tests were within normal ranges.

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2. Associate Professor of Periodontology, School of Dentistry, Georgetown University; Washington, D.C.

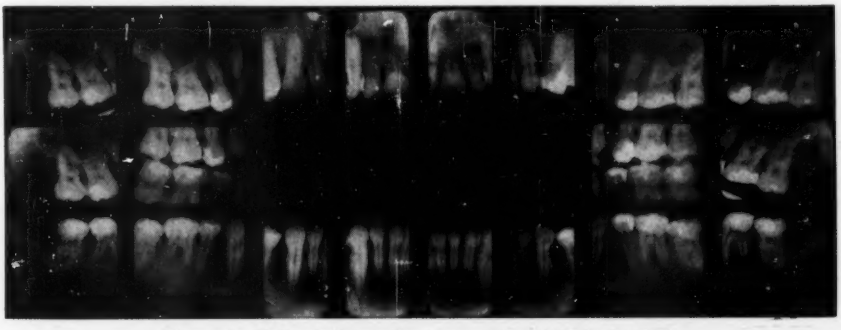

Figure 1. Full mouth roentgenogram taken in October 1953.

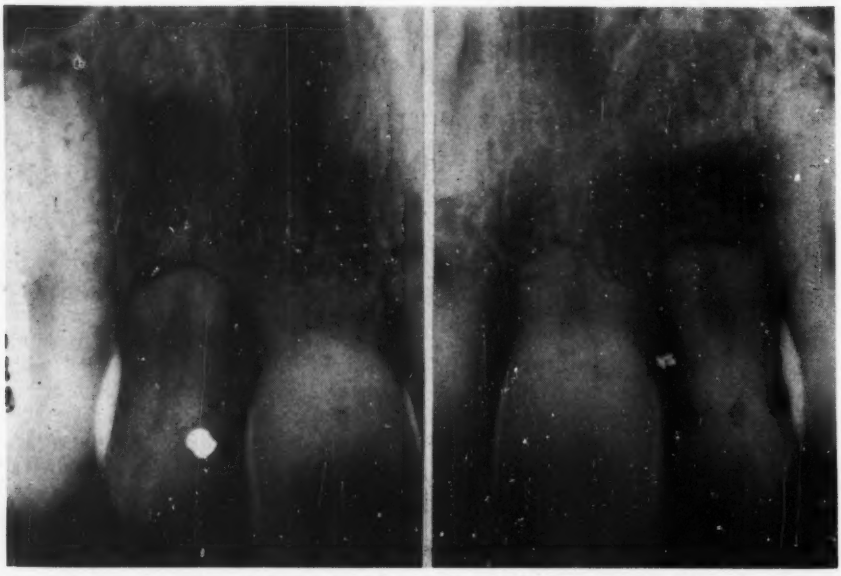

Figure 2. Roentgenograms taken in October 1953. Upper incisor area. Notice the extreme root resorption and the fact that a periodontal space can be made out quite well around the root surface.

Therapy for the marginal gingivitis was executed with good results.

The patient was seen again more than five years later, on December 31, 1958. Marginal gingivitis was again noticeable particularly around the upper right central incisor (Fig. 3). The full mouth roentgenogram taken at this time (Fig. 4) by the same oral roentgenologist employing an identical technique shows the process of root resorption to be continuing only slightly, except in the right upper first molar and the right upper central incisor (Fig. 5), which two teeth show considerable additional resorption; both teeth tested non-vital at this time. Even now though, none of the teeth showed signs of increased mobility. Probing around the sulcus of the involved upper right central incisor indicated a 4-millimeter deep pocket on the mesial side communicating with the area of root resorption and the peri-"apical" area. The patient's generalized marginal gingivitis was re-treated, and cleared up promptly with exception of the area around the upper right central incisor; extraction of this non-vital tooth and of the non-vital right upper first molar was refused by the patient. When these teeth will be extracted it is planned to examine them histologically.

Figure 3. Clinical photograph taken in December 1958. Note gingivitis in the upper and lower anterior sector, particularly (arrow), in the upper right central incisor.

DISCUSSION

A recent good review of the problem of root resorption following orthodontic therapy exists by Phillips.[1] Whether the past experience of the patient with poliomyelitis

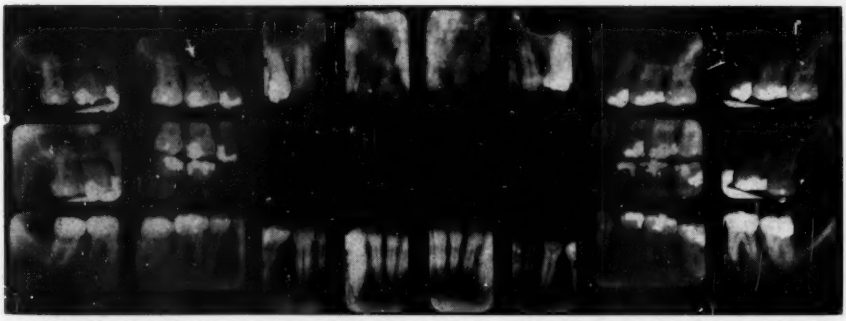

Figure 4. Full mouth roentgenogram taken in December 1958. Note the continuing root resorption on the upper right first molar (arrow), and on the upper right central incisor (arrow). In the other teeth the process of root resorption seems to progress only moderately.

could have had any bearing on the root resorption we do not know; other muscular spasms and occasionally also trismus may be present during the acute phase of this disease. It is known that trismus of the masticatory musculature may initiate root resorption.[2]

Two further points in our case seem to be worth discussing. The one is that with this degree of root resorption no pathologic mobility could be noted. Whether this is due to ankylosis, we do not know. However, the roentgenograms of these teeth show a more or less uninterrupted periodontal space around the remainder of the roots of these teeth. Ankylosis of the buccal or lingual part of these would still be conceivable. No apparent shortening[3] of any tooth in the patient's mouth could be noted, further placing in doubt whether ankylosis exists. It would also be well to recall that a few millimeters of root surface periodontal attachment near the crest might well account for the clinical firmness of these teeth. It has been noted before that due to the larger surface involved many more fibers of the periodontal ligament insert themselves *per millimeter of root length* into this more cylindrical area of the cementum, than to the approximately cone-shaped apical part.

Figure 5. Roentgenogram taken in December 1958. Notice the continuing resorption in the upper left central incisor (external and/or internal resorption).

Secondly we wish to point out the possibility that localized, apparently marginal gingivitis may be caused by a process of extreme root resorption combined with loss of pulpal vitality.

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Simultaneous Healing of a Treated Infrabony Pocket and Alveolus

by JOE H. SMITH, D.D.S., B.S.D., *Dallas, Texas*

THE phenomenon of fibrous reattachment in the infrabony pocket is an accepted fact by most periodontists and general practitioners in dentistry today. However, radiographic evidence for most successful attempts is subject to criticism, because of the variables which can influence the image on the radiographic film.

Friedman[1] has critically evaluated the criteria as presented in the clinical and experimental evidence for reattachment that has appeared in the literature. He, also, suggests that following an attempt at reattachment there may be a better quality of bone present instead of a greater quantity. Cahn[2] presented two cases which tend to confirm this possibility.

The case presented here affords the opportunity of comparing the regrowth of bone in an infrabony pocket following therapy with that of an alveolus of an extracted tooth adjacent to it.

The patient, a white man, age 44, presented for periodontal consultation 1-3-58. He was edentulous distal to the LR 4 and LL 4. There was a generalized recession of alveolar bone around the remaining teeth as a result of long standing, chronic inflammation. The gingiva were firm and fibrotic and heavy deposits of supra and sub gingival calculus were present. There was a 10mm. infrabony pocket on the mesial surface of the LL 3. Because of the poor prognosis of the lateral incisors, it was deemed advisable to extract them and construct an acrylic splint from LR 4 to LL 4 replacing the lateral incisors prior to periodontal surgery.

The teeth were extracted on 1-14-58 and the splint seated temporarily at that time. The next day the patient presented for periodontal surgery at which time the radiograph in Fig. 1 was made. He was premedicated with 20 cc. of elixir Butisol Sodium.

Infiltration anesthesia was used around the remaining teeth. An incision was made to the depth of the gingival pockets and the tissue excised. Because of the lack of sufficient attached marginal gingiva remaining, an extension of the vestibular trough was made. This afforded good access and the infrabony pocket was curetted for removal of all soft tissue. The root surface was planed to remove all hard deposits.

Following completion of the surgical procedure a strip of Telfa was covered with a thin layer of surgical pack and folded. This was placed over the infrabony pocket to prevent the pack from going into the bony defect. Antibiotic therapy was started and maintained for three days. Routine dressing for the rest of the surgerized area was applied. The pack was changed at weekly intervals until three weeks had elapsed from day of surgery. By this time, granulations had covered the bone completely. Routine post operative care was followed by the patient.

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Figure 1

The site was examined with the periodontal probe and radiographs six months later. Fig. 2 shows the radiograph made at that time. There was a three mm. soft tissue pocket remaining. It has been two years since the time of surgery and there have been no untoward changes. Permanent splinting was used to replace the temporary splint, and a partial denture was constructed to replace the missing posterior teeth.

DISCUSSION

Obtaining successful reattachment in an infrabony pocket is based primarily on the phenomenon of repair. It is essential to have three remaining walls of bone as advocated by Prichard,[3] and a fairly deep, narrow pocket is considered more favorable than a shallow broad pocket. Also, as Goldman[4] has pointed out, it is essential to

Figure 2

remove all the soft tissue lining the pocket including the remaining transseptal fibers. To make sure all the transseptal fibers are removed it is necessary to contact the bone at the base of the pocket. The bone should not be unduly traumatized, but on the other hand, it is wrong to leave any intact transseptal fibers. This slight injury to the bone does not cause any permanent damage, and indeed will contribute to the repair bio-mechanism. McClean and Urist[5] have stated, "New bone formation is an automatic reaction to *any* form of injury to bone tissue. The injury and reaction are so much a part of each other that the damaged tissue seems to introduce local factors which produce osteogenesis."

In this "surgical curettage" of the infrabony pocket, a situation is produced similar to that caused by the removal of a tooth, and in the healing process of the wound new bone and a new periodontal membrane are produced. If there is no interference to the healing process, the infrabony defect will heal to the level of the remaining alveolar bone the same as in an extraction wound.

Inasmuch as the blood clot is the physiological dressing for the bone, it should be afforded protection for three or four days until the granulations cover the exposed bone. The two most common causes for premature loss of the blood clot are (1) the patient rinsing and dislodging it, and (2) through bacterial action.[6] Because the clot is protected by the pack from inadvertent dislodgment, we need only to be concerned with the bacteria and their enzymes. The strepto-coccus viridans is one of the most common pathogens in the oral flora and is capable of causing fibrinolysis. It is the most common bacteria associated with bacteremia following oral surgical procedures. Because the incidence of bacteremia[7] following periodontal surgery has been shown to be as high as 25%, it is justifiable to use antibiotics prophylactically, providing there is nothing in the patient's history to contraindicate its use.

CONCLUSION

A favorable result may be obtained in the therapy of an infrabony pocket if proper cases are selected, thorough curettage of the pocket is affected making certain the intact transseptal fibers are removed and a favorable environment is provided for the wound during the repair process. The stimulus for new bone formation is provided by the injury to the remaining bone just as it is in an extraction wound.

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Book Reviews . . . .

Seldom do two new books dealing with similar subjects appear at the same time. Seldom is one asked to review two books at the same time. These are important texts dealing with a difficult and important subject, and their differences need critical evaluation. Whereas Dr. Schwartz emphasizes the important and sometimes serious emotional predisposition of patients suffering with the more common temporomandibular joint disorders, and the need for correction of same prior to (and many times without) manipulative care of the occlusion, Dr. Shore emphasizes, without adequate scientific support, numerous ill-effects supposedly caused by occlusal abnormalities, and devotes most of his text to his techniques of occlusal equilibration.

DISORDERS OF THE TEMPOROMANDIBULAR JOINT—Diagnosis, Management, Relation to Occlusion of Teeth. Written by Laszlo Schwartz, D.D.S., Clinical Professor of Dentistry, Columbia University; Director, Temporomandibular Joint Clinic, Columbia-Presbyterian Medical Center and eigtheen contributors. *W. B. Saunders Co., Philadelphia and London, 1959.* 471 pages; 458 illustrations on 168 figures, with an Introduction by Charles Ragan, M.D., Professor of Clinical Medicine, Columbia University, and a Foreword by Harry Sicher, M.D., D.Sc., Professor of Anatomy, Loyola University School of Dentistry, Chicago. Illustrated by Robert J. Demarest. *Price: $15.00.*

This is an excellent text, carefully compiled by a student of this field with the help of recognized contributors from the fields of dentistry and oral surgery, physical medicine, neurology, surgery, anatomy, psychiatry, neurosurgery, anesthesiology, radiology, and otolaryngology. Dr. Schwartz, in the preface, states that "One hope, and possibly the most basic, is that the concepts, diagnostic procedures and therapeutic methods here presented, having as they do the virtue of simplicity, will be widely practiced. Physiologic and psychologic methods should supersede the mechanical technics that have dominated the management of temporomandibular joint disorders during the past quarter of a century." He makes a plea for closer cooperation among dentists and physicians in the diagnosis and management of temporomandibular joint disorders. The book is testimony to the fact that such cooperation has been found. It is divided into 5 sections: general considerations, diagnostic procedures, differential diagnosis, methods of treatment, and management of disorders. These sections are broken into 37 chapters. Dr. Schwartz is the sole author of 16 and co-author of 1. The other 20 chapters are written by contributors from the fields mentioned above. Among other things, this book is concerned with the study of the findings in over 2,500 patients seen at the Temporomandibular Joint Clinic. Occasional cases were associated with osteo-arthritis, rheumatoid arthritis, mandibular fractures and neoplasms involving the area. The majority of cases were described as a pain-dysfunction syndrome associated with a dull ache, limited movement, clicking, subluxation and especially, painful muscle spasms. It is believed that what the patient does with his jaws in reacting to the stress of life is more important than any malocclusion that he may have, no matter how irregular. It was noted that changes in occlusion accomplished by a dentist by grinding or placing restorations frequently resulted in the initiation of this syndrome. He believes that the broad approach to the problem as presented, "—cannot help but free us from the belief that the occlusion of the teeth is always stable and cannot change in the same individual from one moment to the next." "Our investigations have demonstrated that though 'occlusal interferences' or 'premature contacts' may contribute to the onset of muscle spasm, muscle spasm can and does create 'interfer-

ences.' Our research has made us much more hesitant in undertaking treatment involving occlusion. We feel that it should not be undertaken until both the patient and the muscles are relaxed." "We are learning to view the patient as a whole, and if we feel reservations regarding treatment we do not brush them aside just because we see an occlusion that offends our sense of what is 'normal'."

Throughout the text, emphasis is placed on the underlying tensions and the emotional status of such patients with associated clenching and grinding of the teeth. Treatment of the underlying causes for such "tensions" and treatment of the results of such tensions (bruxism) are emphasized, and treatment by occlusal alterations is de-emphasized. Entire chapters are devoted to such things as the occlusion of the teeth; electromyography; pain of various regional origins; and treatment techniques including therapeutic exercises; local anesthetics; muscle relaxants; and psychology. No little space is devoted to causes of complaints resulting from the use of certain techniques of treatment advocated in the past.

In the reviewer's opinion this text is a monumental contribution and it is recommended without reservation.

OCCLUSAL EQUILIBRATION AND TEMPOROMANDIBULAR JOINT DYSFUNCTION, by Nathan Allen Shore, D.D.S., Instructor in Occlusal Equilibration and Temporomandibular Joint Dysfunction in the Post-Graduate Schools of the 1st, 2nd, 3rd, and 10th District Dental Societies of the New York State Dental Society; Dentist to Out-Patients, Department of Surgery (Oral Surgery), the New York Hospital; a Founder, Past-President and Fellow of the Society of Oral Physiology and Occlusion. 242 Illustrations. *J. B. Lippincott Co., Philadelphia and Montreal. Price: $12.50.*

This author has made an earnest attempt to cover a rather broad field and the results are confusing, contradictory and sometimes non-informative. As the title implies, he is first and foremost concerned with occlusal equilibration. The book is broken into 14 chapters, 5 of which have both the words "equilibration" and "occlusion" in their titles, and 2 more of which have "occlusion" alone. The emphasis of the text is placed on the "continued noxious stimulation" due to existing minor occlusal abnormalities and interferences which the author assumes to be the cause of most temporomandibular joint disorders and implies that same are important in many other symptoms and forms of pathology. "—if there are interfering occlusal contacts, this orderly and integrated (stomatognathic) system is disrupted, and abnormal jaw movements, disease, degeneration and destruction inevitably result." Dr. Shore variously says or implies that malocclusion can be the cause of the following: pulp stones, hyperemia, pulpitis, and necrosis; loss of the lamina dura, increase in periodontal membrane width, cemental tears, resorption, and apposition of same; tears in the epithelial attachment, deepening of periodontal pockets, gingival recession and hypertrophy, epithelial nodules, traumatic crescents, gingival festoons and clefts; linear depressions and distended mucosal veins; pain throughout the head including the nuchal area and sometimes extending to the finger tips; "periapical osteofibrosis"; edema, swelling, and pain of the infra-temporal fossa, the major salivary glands, and regional lymph nodes; metallic taste aberrations and other signs and symptoms.

Thus, without proof or careful consideration, he lays at the door of malocclusion a vast number of unrelated disorders, according to current concepts. An example of the scientific writing follows: "In patients who have weak and hypermobile joints, breakdown results in temporomandibular joint syndrome. However, if the temporomandibular joint is strong enough to resist breakdown, the periodontium supporting the teeth will give

way. Proof of the fact that the temporomandibular joint is stronger than the periodontium lies in the truth that, at the age of 80, all human beings still possess their temporomandibular joints, while few of them have good teeth supported by a strong periodontium." (!)

Thus, this text appears to be adequate in demonstrating to the dentist the author's techniques of equilibrating and otherwise modifying the occlusion. The value of this procedure in the prevention and treatment of the many disorders, including "temporomandibular arthrosis" is questioned. Ten case histories are furnished. *D. F. M.*

OPEN DOOR TO HEALTH. By Fred D. Miller, D.D.S., as told to Edna Yost, A.B., Litt. D. 179 pages, indexed, published by *Devin-Adair Company, N. Y. C. 1959. $3.95.*

Dr. Frederick D. Miller in 1912 while at the University of Pennsylvania Dental School was deeply impressed by a statement made by Dr. Kirck, the dean. Dr. Kirck, the father of five children said: "Our children had fine teeth while living at home where we had control of their food habits, but soon after leaving home they all developed bad teeth." This thought planted a seed in Dr. Miller's mind which has been nourished over the subsequent years by observation and intensive study and has finally blossomed in the form of a remarkable book.

The dentist is in a unique position as far as patient education is concerned. In the first place he sees most of his patients regularly, secondly he has a splendid opportunity to detect early signs of degeneration and thirdly he has a "captive audience." This book will assist the dentist in his presentation and should be recommended reading for all patients interested in better health and subsequently better teeth and supporting tissues.

Dr. W. Ward Tracy

TUFTS UNIVERSITY

DPG. 709—PROSTHETICS FOR THE PERIODONTAL PATIENT

May 9-12, 1960

by DR. IRVING GLICKMAN, PROFESSOR AND CHAIRMAN OF THE DEPARTMENT OF ORAL PATHOLOGY AND PERIODONTOLOGY, DR. A. ALBERT YURKSTAS, ASSOCIATE PROFESSOR OF PROSTHETIC DENTISTRY, DR. FRANK A. EICH, ASSISTANT PROFESSOR OF PROSTHETIC DENTISTRY (Crown and Bridge), DR. BENJAMIN PATUR, ASSISTANT CLINICAL PROFESSOR OF PERIODONTOLOGY, DR. R. SHELDON STEIN, LECTURER IN POSTGRADUATE PROSTHETICS

When prosthesis is required as part of the overall treatment of periodontal disease, it must be designed so as to insure retention of the natural dentition. This course was arranged for the purpose of teaching how prosthesis should be constructed to provide maximum benefits to the periodontium. It will be presented by lectures, closed-circuit television, motion pictures and seminars. The curriculum, arranged by clinicians experienced in the management of combined prosthetic-periodontal problems, is comprehensive and practical. The following is a cross-section of the subjects which will be covered: Periodontal splints, indications and contraindications for fixed and removable prosthesis in the treatment of periodontal disease, and the reorientation of functional forces with prosthesis. Tuition $150.00.

OFFICERS OF CALIFORNIA ACADEMY OF PERIODONTOLOGY 1960

Newly elected Officers of The California Academy of Periodontology:—

Front row, left to right: Clarence E. Butler, Treasurer, Jean P. Cantou, Immediate Past President, B.O.A. Thomas, Second Vice President, J. James Pfister, President, Elmer G. Smith, Financial Secretary.

Back row, left to right: Frederick S. Warford, Third Vice President, Stanley S. Sepich, Corresponding Secretary, Mario W. Pieri, Recording Secretary, Antonio R. Sanchez-Corea, Editor, Academy Review, Robert E. Oslar, First Vice President, Lowell N. Peterson, Historian and Librarian.

WESTERN RESERVE UNIVERSITY

Western Reserve University announces a postgraduate course in "Current Periodontal Therapy" June 2, 3 and 4, 1960 by Dr. Henry M. Goldman. The course will include discussions of diagnostic data in periodontal manifestation and evaluation, treatment planning, initial therapy, curettage and gingivectomy, gingivoplasty, osteoplasty, vestibular surgery, local environmental factors influencing therapy. The fee for the course is $100.00.

For further information write Director of Postgraduate Education, School of Dentistry, Western Reserve University, Cleveland 6, Ohio.

ANNUAL BERKSHIRE CONFERENCE

Dr. Charles E. Stuart of California, noted authority in the field of occlusion will be principal lecturer at the Tufts 11th Annual Berkshire Conference in Oral Pathology and Periodontology to be held June 19-23, 1960 in Lenox, Mass. Dr. Stuart has had extensive experience as a leader in his field.

He has been postgraduate instructor at the Gnathological Seminar, University of Oregon, and has also been postgraduate instructor at the University of California, Illinois, Southern California and Emory University. He has directed several research groups in California in the study of occlusion. At the Berkshire Conference Dr. Stuart will devote two days to the following subjects:

Principles and Use of Diagnostic Instruments for Instituting a Therapeutic Occlusion

Principles and Objectives of Therapeutic Occlusion

The Technique for Restoring Occlusion for Patients with Periodontal Problems

PRIZE DR. h.c. RENÉ JACCARD

We have the pleasure of bringing to your attention that the "Prize Dr. h.c. René Jaccard" will be attributed for the third time during the XVI. Congress of The "ARPA Internationale" in Vienna (September 17-22, 1960).

The conditions of candidature for the prize are as follows:

a) Any original scientific work concerning periodontal disease published or performed between 1958 and 1960 can be sent in.

b) It is not necessary for the author to be a member of the ARPA.

The proposed papers for candidature shall be sent, by registered post, in six exemplars, to the president of the "ARPA Internationale" (Prof. A. J. Held, Institut de médecine dentaire, 30 rue Lombard, Geneva, Switzerland) at latest the 15th of May 1960.

The prize (or prizes) shall be delivered during the XVI. Congress of the "ARPA Internationale," in Vienna.

UNIVERSITY OF PENNSYLVANIA

The University of Pennsylvania School of Dentistry announces a one-day Refresher Course in Periodontal Therapy by Dr. Henry M. Goldman and Dr. D. Walter Cohen on May 14, 1960. This seminar will review current advancement in Periodontal Therapy for individuals who have attended a postgraduate course in Periodontal Therapy at this institution or at Beth Israel Hospital, Boston, Mass. Fee will be $15.00.

For further information write Director, Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce Street, Philadelphia 4, Pennsylvania.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

Tufts University School of Dental Medicine announces a postgraduate refresher course in "The Diagnosis and Treatment of Pain and Other Symptoms Arising from Temporomandibular Joint Disturbances" Tuesday and Wednesday, May 24 and 25, 1960. Class will be limited; tuition is $50.00.

For additional information write Director, Graduate Division Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston 11, Massachusetts.

Letters to the Editor . . . .

Dear Sir:

I am pleased with the interest aroused by the article, "Papillon-Lefevre Syndrome: Precocious Periodontosis with Associated Epidermal Lesions," published in the July 1959 issue of the *Journal.*

Since the article was published Dr. Rosa Iusem Carvel[1] of Loyola University, New Orleans, has sent a reprint of a thesis she published in Buenos Aires dealing with two sisters with a similar problem—overwhelming alveolar destruction with associated epithelial lesions. None of the authors on this subject have quoted Dr. Carvel's work, for only recently has a Spanish Language Cumulative Index become available.[2]

Dr. Walter Cohen has also brought to my attention an article by Greither[3] which reports two similar cases, and lists a total of 29 cases from the European and American literature. The two cases from Carvel and the one case I reported are not listed by Greither, so these three cases would bring the known international total to 32 cases.

I was especially interested in the letter by Dr. John Oppie McCall in the January 1960 issue of the *Journal,* challenging the diagnosis of periodontosis as the etiologic factor in the advanced drifting of the right maxillary central incisor of the case I reported. Dr. McCall was certain the tooth had been involved in a traumatic accident.

The best answer to this question I could give Dr. McCall would be to relate the telephone conversation at the time the boy was referred.

The patient was referred by an out of town dentist who stated he had heard me lecture on replantation of avulsed teeth. He had a patient in the office with a maxillary central incisor "sticking straight out and ready to fall out," and could I replant it?

I of course asked how long ago the accident happened and the dentist replied, "That's a funny thing; the youngster denies ever being hit in the mouth, and his mother who is here with him says that's right, that the tooth has slowly drifted into this position over a period of two years. Of course they both are lying."

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1. Iusem, Rosa. Dos casos de grave reabsorcion alveolar en la infancia. Buenos Aires, Sebastian de Amorrortu e Hijos, 1945. (19 pp.)

2. Muruzabal, Margarita, et al. Indice de la literatura dental periodica en castellano y portugues. Buenos Aires, Asociacion Odontologica Argentina, 1956. (132 pp.)

3. Greither, Von A. Keratosis palmo-plantaris mit periodontopathie (Papillon-Lefevre). Dermatologica, 119:248-61, November, 1959.

Suspecting periodontosis, I asked the dentist to return to the patient and probe the mesial surfaces of all four first molars. He returned to the phone and said, "I'll be damned, how did you know those pockets were there?"

I agree with Dr. McCall, this case is not a true picture of juvenile periodontosis. The second case described and pictured in the article, as reported by Schluger, is more typical of these cases with the overwhelming destruction and, as Dr. McCall points out, the greatest alveolar resorption around the mandibular incisors.

I also erred in not reporting the pulp test of this tooth specifically. It was vital when presented and remained vital until the day it was extracted.

John I. Ingle, D.D.S., M.S.D.
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To the Editor:

The article Histogenisis of the Gingival Sulcus Epithelium in the Rat by Uohara in the October number of the Journal was interesting. Dr. Uohara has done a very good piece of work on this subject. I note that he found himself unable to come to a definite conclusion as to the possible attachment of the epithelium to the enamel as eruption of the tooth progressed. The difficulty centers in the fact that enamel becomes decalcified during preparation of the histological specimen.

Under the circumstances I thought it might be worth while to cite some clinical observations bearing on that point. During the time I was Director of the Guggenheim Dental Clinic I was frequently asked to examine children having gingival inflammation. This occasionally was found on the labial gingivae of permanent upper incisors, especially the central incisors. It occurred when these teeth were incompletely erupted, which meant that their crowns were partially covered by the gingiva. Invariably I found in these cases that the gingivae had become detached from the enamel nearly up to the cemental-enamel junction. On the contrary, in teeth at a similar stage of eruption but without perceptible gingival inflammation, there was no space between gingiva and enamel. That is, in these cases I was unable to pass even a fine probe into the gingival sulcus for more than one or two millimeters except by using considerable force. There was obvious attachment of gingiva to tooth, and it also obviously was this attachment that prevented the onset of gingival inflammation. Dr. Uohara's observations do not say that such attachment could not be. I offer this statement for what it may be worth.

John Oppie McCall

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